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EDITORIAL

THE DOCTOR AS A CITIZEN

The concept of professional secrecy has been a source of ethical contention ever since Hippocrates propounded it. Is a doctor bound to remain silent about things of which he is aware as the result of his attendance on a patient, if silence will, for instance, imperil the lives of others or the proper functioning of the machinery of State? Frequently his duty as a doctor conflicts with his duty as a citizen.

The Hippocratic Oath runs: 'I will keep silence regarding that which, within or without my practice, I shall see or hear in the lives of men which should not be made public, holding such things unfit to be spoken.' This leaves the decision a matter of interpretation or of opinion. The World Medical Association's International Code of Ethics has it: 'A doctor owes to his patient absolute secrecy on all which has been confided to him or which he knows because of the confidence entrusted to him.' This is more explicit.

The British Medical Association's official view is much more rigid: 'A practitioner shall not disclose voluntarily, without the consent of the patient, preferably written, information which he has obtained in the course of his professional relationship with the patient. This includes information concerning criminal abortion, venereal disease, attempted suicide (a crime in Great Britain), and concealed birth. The State has no right to demand information except where notification is required by statute, such as in infectious disease.'

In order to form an idea of the average man's outlook on the point, a British doctor recently sent the following questions to about 100 doctors and 100 laymen, chosen at random but according to vocation, and analysed the replies he received:[1]

Question 1. A doctor diagnoses epilepsy in the driver of a main-line passenger train. The patient refuses all advice, and says that he will continue to drive trains. Is it the doctor's duty to ignore his patient and report the matter to the police?

Question 2. A doctor attending a woman for abortion finds that it was criminally induced, and is told the name and address of the criminal abortionist. The patient forbids the doctor to report the matter to the police. Is it his duty to ignore this and report it anyway?

Question 3. A doctor treats a worker for hernia. Later the man is injured at work, and fraudulently and success-

VAN DIE REDAKSIE

DIE GENEESHEER AS BURGER

Vandat Hippokrates die konsep van professionele geheimhouding geformuleer het, het dit 'n etiese stryd-vraag gebly. Moet 'n geneesheer die swye bewaar oor dinge wat hy in die beoefening van sy beroep te wete kom as dit byvoorbeeld die lewes van andere, of die behoorlike werking van die staat, in gevaar stel? Dikwels bots sy pligte as geneesheer met sy pligte as burger.

Die Hippokratiese Eed lui: 'Ek sal die swye bewaar oor wat ek binne of buite my praktyk sien of hoor in die lewens van mense, wat nie geopenbaar behoort te word nie, en ek sal dit beskou as ongepas om oor sulke dinge te praat'. Dit laat derhalwe die beslissing aan eie vertolking of mening oor. Die Internasionale Etiese Reëls van die Wêreld- Mediese Vereniging lui as volg: 'n Geneesheer is aan sy pasiënt absolute geheimhouding verskuldig aangaande alles wat in vertroue aan hom meegedeel is of wat hy te wete kom as gevolg van die vertroue wat in hom gestel is'. Dit is duideliker.

Die amptelike mening van die *British Medical Association* is heelwat strenger: ,'n Geneesheer moet nie sonder die toestemming van sy pasiënt, verkieslik skriftelik, vrywilliglik feite aan die lig bring nie waarvan hy kennis opgedoen het as gevolg van sy professionele verhouding tot sy pasiënt'. Dit behels inligting oor kriminele vrug-afdrywing, geslagsiekte, selfmoordpoging (wat 'n misdaad in Brittanje is) en verbergde geboorte. Die Staat het nie die reg om inligting te eis nie, tensy dit deur statuut vereis word, soos in die geval van aansteeklike siekte'.

Ten einde 'n idee te kry van die gewone man se opvatting het 'n Britse geneesheer onlangs die volgende vrae aan ongeveer 100 geneeshere en 100 leke gestel. Hulle is blindweg gekies en die antwoorde ontleed.[1]

Vraag 1. 'n Geneesheer diagnoseer dat die drywer van 'n hooflyn-passasierstrein aan vallende siekte ly. Die pasiënt verwerp alle advies en beweer dat hy nie die werk sal opgee nie. Is dit die geneesheer se plig om sy pasiënt te veronagsaam en die saak aan die polisie te rapporteer?

Vraag 2. 'n Geneesheer behandel 'n vrou vir vrug-afdrywing—'n kriminele geval—en die naam en adres van die afdrywer is aan hom verstrek. Die pasiënt verbied die geneesheer om die saak aan die polisie te rapporteer. Is dit sy plig om dit desnieteenstaande te rapporteer?

Vraag 3. 'n Geneesheer behandel 'n arbeider vir 'n breuk. Die man is later in sy werk beseer en op bedrieg-

fully claims compensation in respect of the hernia, which the doctor knows was neither caused nor aggravated by the injury. Is it his duty to report this to the authorities?

Question 4. In the course of a visit a doctor notices by chance some jewellery corresponding to the newspaper description of property recently stolen in a housebreaking raid. Is it his duty to report the matter to the police?

The answers to these questions were as follows (the figures are approximate):

		Yes	*No*
Question 1 (epilepsy)	Doctors	84	12
	Laymen	82	15
Question 2 (abortion)	Doctors	51	47
	Laymen	55	39
Question 3 (hernia)	Doctors	34	64
	Laymen	47	48
Question 4 (jewellery)	Doctors	34	64
	Laymen	47	49

Analysis of the answers shows an interesting divergence of outlook, based upon moral interpretation rather than upon profession (the legal aspect is not considered here). Thus it is found that:

Over 80% of doctors thought that the engine-driver's epilepsy should be reported, even against his wishes, one-half were in favour of reporting the abortionist, and only one-third favoured positive action in connexion with the fraudulent workman or the jewellery thief.

These figures seem to suggest that the average medical man is only prepared to break his obligation of secrecy where there is a real danger to other persons' lives or health. When it is merely a question of fulfilling his obligations to the State as a citizen he is not prepared to speak. Some critics would probably interpret this to mean that the obligation of secrecy is like the McNaghten rules—to be applied only when thought necessary—and therefore a very comfortable loophole for the medical profession.

However, there is as much to be said in favour of the overriding duty of a doctor as a citizen of his State, as there is to be said for the British Medical Association's rigid view of the secrecy obligation. The two arguments are succinctly summarized by the following passages:

'It is a moral responsibility not to condone crime or fraud; the confidence of the doctor must presume good faith; the patient who acts immorally forfeits his doctor's confidence.'

'The sanctity of private trust overrides that of public trust. I would rather betray my country than my friends.'

1. Dawson, E. C. (1954): Brit. Med. J., **2**, 1474.

like wyse slaag hy in 'n eis om skadevergoeding ten opsigte van die breuk. Die geneesheer weet dat die breuk nie deur die besering veroorsaak of vererger is nie. Is dit sy plig om die feit aan die owerhede mee te deel?

Vraag 4. Tydens 'n besoek sien 'n geneesheer, bloot by toeval, juweliersware wat klop met die koerantbeskrywing van eiendom wat onlangs as gevolg van 'n huisinbraak gebuit is. Is dit sy plig om dit aan die polisie te rapporteer?

Die volgende antwoorde is verkry (die syfers word by benadering gegee):

		Ja	*Nee*
Vraag 1 (vallende siekte)	Geneeshere	84	12
	Leke	82	15
Vraag 2 (vrugafdrywing)	Geneeshere	51	47
	Leke	55	39
Vraag 3 (breuk)	Geneeshere	34	64
	Leke	47	48
Vraag 4 (juweliersware)	Geneeshere	34	64
	Leke	47	49

Ontleding van die antwoorde toon 'n interessante meningsverskil, die grondslag waarvan 'n morele vertolking en nie 'n beroep is nie. (Die regsaspek word nie hier oorweeg nie). Dus is die bevinding dat:

Meer as 80% van die geneeshere die mening toegedaan is dat die drywer se vallende siekte gerapporteer moet word, selfs teen sy wense; 50% was ten gunste daarvan om die afdrywer te rapporteer en slegs een-derde was van mening dat stappe teen die bedrieglike arbeider en die dief geneem moet word.

Hierdie syfers skep die indruk dat geneeshere oor die algemeen slegs hul plig om die swye te bewaar sal veronagsaam as die lewes of gesondheid van ander op die spel is, maar as dit net 'n kwessie van burgerlike plig is, is hy nie bereid om te praat nie. Sommige kritici sal waarskynlik dit vertolk dat die plig van geheimhouding net soos die McNaghten reëls, net toegepas moet word as dit nodig is.

Daar is egter net soveel ten gunste van die stelling dat die plig as burger swaarder moet weeg dan die plig as geneesheer as vir die *British Medical Association* se strenge opvatting van die plig van geheimhouding. Die volgende aanhalings gee 'n bondige opsomming van die twee gesigspunte:

'It is a moral responsibility not to condone crime or fraud; the confidence of the doctor must presume good faith; the patient who acts immorally forfeits his doctor's confidence.'

'The sanctity of private trust overrides that of public trust. I would rather betray my country than my friends.'

1. Dawson, E. C. (1954): Brit. Med. J., **2**, 1474.

5-HYDROXYTRYPTAMINE

This substance, also referred to in the literature as serotonin and enteramine, is an indole derivative. It has long been known as a naturally-occurring substance. In recent years it has been isolated, identified and synthesized, and has recently been shown to be of clinical significance.

It is almost a hundred years since it was demonstrated that the vasoconstrictor action of blood increases when it clots. However, it is only in recent years, in the investigations of the humoral factors involved in arterial hypertension, that the fraction called serotonin was isolated from blood serum.[1] Enteramine, shown to be identical with serotonin, was first discovered by relatively crude methods as an active substance in the mucosa of

the rabbit's stomach; it was regarded as an amine originating from 'enterochromaffin or argentophile cells' in the gastro-intestinal tract of mammals—hence the name enteramine.

The serum vasoconstrictor has long been regarded as originating from platelets, but this is apparently not correct. It is held that the substance aids in haemostasis, but this problem needs more study. Now that anti-serotonins and synthetic serotonins are available investigations are proceeding in many laboratories, and the role of the substance in myocardial infarction, vascular injury and embolism is being studied.

The finding of the similar substance (enteramine) in the intestine has suggested that it controls gastro-intestinal activity. It seems that 5-hydroxytryptamine is secreted by the argentaffin cells in the intestine and has a stimulating effect on intestinal movements.

The platelets, whose high content of 5-hydroxytryptamine led to isolation of the substance, do not elaborate the hormone but take it up as they do histamine and other substances; these are presumably released under certain conditions, such as at the site of an injury.[2]

The syndrome which includes argentaffinoma, pulmonary stenosis and transient macular cyanosis [3–5] would appear to be established as an entity. The vascular lesions seem to develop from hypersecretion of 5-hydroxytryptamine—a 'new' hormone secreted in excess by an endocrine tumour, the argentaffinoma.

The pharmacology of this newly-discovered hormone is being investigated in many centres. It has a constricting action on smooth muscle generally and in some species it produces systemic hypertension. Not enough is known regarding its action on the pulmonary circulation, and difficulty is encountered in explaining the production of pulmonary valvular stenosis so definitely present in the syndrome. In man local congestion and venous spasm follow intradermal injection of the hormone.[2]

The hydroxytryptamine content of the serum of many animal species has been investigated and it seems justifiable to postulate its identity with a stable non-pituitary antidiuretic substance.[6] It apparently originates in serum during coagulation. Work is in progress to determine what changes occur in the content of 5-hydroxytryptamine under various experimental and pathological conditions.

The estimation of hydroxytryptamine can be carried out by biological assay, but more precise chromatographic methods are available. The development of a suitable procedure for estimating the breakdown products such as 5-hydroxy-indole-acetic acid, which is excreted in the urine, will provide another method of investigation. With such tests it may become easily possible to diagnose hyperactivity or tumours of the argentaffin cells biochemically before advanced clinical signs appear.[7]

1. Page, I. H. (1954): Physiol. Rev., **34**, 563.
2. Reid, G. and Rand, M. (1952): Nature, **169**, 800.
3. Thorson, A. *et al.* (1954): Amer. Heart J., **47**, 795.
4. Editorial (1954): Lancet, **2**, 372.
5. Pernow, B. and Waldenström, J. (1954): Lancet, **2**, 951.
6. Erspamer, V. and Sala, G. (1954): Brit. J. Pharmacol., **9**, 31.
7. Editorial (1954): Lancet, **2**, 958.

THE EFFECT OF MELADININ IN THE TREATMENT OF VITILIGO*

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Vitiligo or primary acquired leucoderma is a condition which has been recognized for centuries, although probably in the past there has been considerable confusion between it and the various forms of secondary leucoderma. There is no need in this paper to give a detailed clinical description of the disease nor is it intended to go into the biochemistry of melanogenesis beyond to state briefly that melanin is formed by the enzymatic oxidation of tyrosine by tyrosinase, and the site of this reaction is the dendritic-shaped melanocytes lying at the epidermo-dermal junction. It has been suggested by Lerner and Fitzpatrick [1] that pigment-formation is controlled by adrenal hormones which through the pituitary-adrenal axis are secreted to inhibit the release of a pigment-hormone from the pituitary. This melanocyte-stimulating hormone (M.S.H.) has been shown in experimental work to cause temporary hyperpigmentation in normal human beings. It has not as yet been possible to relate these findings to vitiligo, and microhistological studies of the skin in this condition reveal no organic change in the melanocytes.

Although numerous hypotheses have been suggested to explain the aetiology of vitiligo, there has as yet been no supporting evidence to confirm these. As a result of clinical observations many workers have attributed the condition to some nervous shock in the form of a stress syndrome, and this might be supported by the suggestion of a pituitary-adrenal axis, but it is difficult to explain the patchy yet clear-cut distribution of the depigmented lesions.

In the past numerous treatments have been advocated, from the various forms of light therapy in use 50 years ago to the more recent use of gold sodium thiosulphate

* Based on a paper by Dr. Leeming presented at the South African Medical Congress, Port Elizabeth, June 1954.

by Lindsay in 1929 [2] and its combination with 10% oil of bergamot and phototherapy by Burgess [3] in 1934; more recently thorium X has been suggested, and that overworked panacea for all ills, vitamin-B complex, in conjunction with dilute hydrochloric acid and para-aminobenzoic acid.

A recent report by Katchkovskiy [4] in Moscow (1953) claims that good results have been obtained with ultra-violet rays (U.V.R.) followed by painting with tincture of iodine.

Careful analysis has shown that none of these methods of treatment is likely to give at the best more than a 5% chance of cure, which may well be explained by cases of spontaneous remission, that are sometimes seen.

AMMI MAJUS

For many hundreds of years it has been known in the North African desert that certain extracts of the *Ammi majus* plant have the power of sensitizing the skin to sunlight, as a result of which an acute inflammatory reaction develops, leading frequently to normal repigmentation. But these crude extracts produce in quite a high percentage of cases certain severe side-effects such as abdominal pain, nephritis, cirrhosis of the liver, exfoliative dermatitis and coma. In 1941 Rogad Fahmy and Abou Shady,[5] in Egypt, prepared a soft alcoholic extract of this drug, which was given in enteric capsules. Although it was a definite advance on previous therapy toxic side-effects were still frequent, though less severe, and, as a result of further research by the same workers, 2 crystalline extracts were prepared, viz. ammoidin $C_2H_8O_4$ and ammidin $C_{16}H_{14}O_4$, neither of which exceeded 0.5% of the original powder. El Mofty [6] working with these extracts in 1948 treated a series of 20 cases, using 3 methods, viz. (1) local application of 1% ammoidin in glycerin and alcohol, (2) oral administration in a dose of 0.05 g. daily, and (3) a combination of (1) and (2).

In this series of 20 El Mofty reported 10 complete cures, 6 failures and 4 doubtful reactions, which were certainly most impressive results. The main feature noticed in repigmentation was the development of follicular pigmented islands which coalesced. All failures were treated for at least 3 months before being regarded as such.

Further extraction of the *Ammi majus* led to the isolation of another crystalline extract, majudin, ($C_{12}H_8O_4$), which is also present in large quantities in oil of bergamot.

A further series of 22 cases was reported by El Mofty [7] in 1952, which gave an even higher proportion of successful results.

Sidi and Bourgeois-Gavardin [8] (1953) reported a series of over 100 cases treated over a period of 2 years. They gave 3 or 4 tablets of Meladinin† daily, allowing 8 days' break each month. The paint was applied to all lesions 1 hour before exposure to sun or U.V.R. and the surrounding areas were painted with 10% para-aminobenzoic acid in 60% spirit to protect them. They found that in white skins an acute vesicular reaction prevented satisfactory pigmentation. Best results were obtained by daily short exposures, which caused a gradual repigmentation. Sensitive areas of skin were painted with a diluted solution, either half or quarter strength. Their analysis of cases showed that, out of 84 who persisted with treatment and were followed up, 7 were completely cured, 10 were almost completely repigmented, 17 showed definite improvement, and 50 showed partial repigmentation. They reported no complete failures. They found that children responded more satisfactorily than adults and also that the shorter the history, the better the prognosis.

†'Meladinin' is the trade name of the extract of *Ammi majus* prepared by the Memphis Chemical Co., Cairo.

PRESENT SERIES OF CASES

The series of 20 cases to be discussed in this paper was started in 1953. The cases were selected as showing the typical clinical features of vitiligo. They consisted of 17 Indians, 2 Africans and 1 European. In each case a Wassermann test was performed to exclude syphilis, and the lesions were photographed. All treatment was carried out in the Department of Physical Medicine at King Edward VIII Hospital, Durban.

As a result of a warning in a personal communication from Dr. Louis Forman of London on the strong photosensitizing powers of Meladinin, the initial approach was cautious and this caution appeared to be more than justified in several cases by the acute reaction which developed after initial irradiation.

Initially a paint was applied having the following percentage formula:

Ammoidin	0·75
Ammidin	0·25
Acetone	10
Propylene glycol	10
90% alcohol	79

Thrice weekly this was painted on the lesions and the patient then exposed to the midday sun for about 15—20 minutes; if the reaction was mild U.V.R. was then used. In the 1 European and 7 of the Indians this caused an extremely acute oedematous and vesicular reaction in the areas treated, as a result of which the treatment was modified. This modification consisted of suspending treatment until the reaction had subsided and then renewing with the paint diluted with S.V.R. to form a 25% or 50% solution.

The present series of cases is too short for statistical deductions. Individual case details are given in Table I and the results are summarized in Table II. General impressions are recorded, particularly as experimental trial was necessary to find the best combination of topical and oral administration of the drug and the best type and dosage of irradiation.

As, so far as we know, there have been no previously published results regarding the treatment of vitiligo in the deeply pigmented races, who tolerate vastly heavier dosage of ultra-violet irradiation than do Europeans or lightly pigmented peoples, the details of physical treatment given in the earlier series of cases (see above) were of little assistance.

In general, with non-pigmented patients, the dosage of ultra-violet is based on the development of the earliest perceptible erythema on the untanned skin

TABLE I

Case No.	Race Age and Sex	Duration	Areas Affected	Method of Treatment	Duration of Treatment	Method of Irradiation	Reaction	Signs of Repigmentation	Remarks
1.	I.F.14	3 mths.	Lips and arms	Paint Pills Both	6 wks. 2 wks. 4 mths.	T.W. 6″ 3—6 mins. T.W. 6″ 2—6 mins. No U.V.R.	Oedema and vesiculation of lips (4th degree erythema)	Islands	Complete repigmentation. Acute light sensitization developed temporarily during treatment.
2.	I.M.10	'Years'	Forehead, abdomen, ant. surfaces of both legs	Paint and Pills 1/day	6 mths.	T.W. 6″ 3—30 mins.	4th degree erythema	Islands	Acute vesiculation in leg lesions within 48 hrs. in spite of no treatment with U.V.R. Developed islands of pigmentation on legs which had not been irradiated or painted. Poor response on forehead. Abdomen repigmented.
3.	E.F.15	8 yrs.	Forehead, neck, angles of mouth, axillae	Paint only	1 mth.	Sun 5 mins. daily. No U.V.R.	Acute eczematous eruption	Nil	An extreme acute sensitization to sunlight on local application of paint. Suggestion of early repigmentation but parents stopped treatment.
4.	A.M.24	10 yrs.	Face, neck, L. pectoral area	Paint Paint and Pills (b.d. or t.d.s.)	4 wks. 4 mths.	Hanovia 18″ 3—25 mins.	2nd degree erythema	Nil	No repigmentation.
5.	I.M.21	3 mths.	Circular patches on eyelids	Paint only	2 wks.	Sun ½ hr. after paint		Islands	Completely repigmented within 1 wk.
6.	I.F.2	6 mths.	Both legs below knee	Paint Paint and Pills 1/day	7 wks. 3 mths.	Hanovia 18″ 5—15 mins.		Islands	Completely repigmented.
7.	I.F.13	2 mths.	Lower 3rd right leg	Paint and Pills 1/day	2 wks.	T.W. 6″ 5—10 mins.		Islands	Rapid and uneventful repigmentation.
8.	I.M.22	3 yrs.	Upper and lower lip	Paint and Pills j.b.d.	7 mths.	Kromayer up to 10 mins.	4th degree acute vesiculation		No sign of repigmentation in spite of acute and repeated vesicular reactions in areas treated.
9.	I.F.35	3 mths.	Shin area and between shoulders	Paint and Pills j.b.d.	3½ mths.	Hanovia 18″ or T.W. 6″ 5—25 mins.	2nd degree erythema	Islands	Back area (covered) completely repigmented after 3 mths. No vesiculation or oedema. Little or no signs of repigmentation in legs.
10.	I.F.16	3 yrs.	Outer aspect thighs, deltoid areas, elbows	Paint and Pills j.b.d.	5 mths.	T.W. 6″ 5—20 mins.	2nd degree erythema	Islands	Hyperpigmentation of shoulder areas. Pigmentation started after 1 wk. and complete in 8—10 wks. Lesions on thighs not treated owing to extent of affected areas.
11.	I.F.14	5 mths.	behind L. ear and between shoulder blades	Paint and Pills b.d.	6 mths.	T.W. 6″ 4—20 mins.	3rd to 4th degree erythema	Islands	This patient reacted well initially but failed to continue treatment. When seen 4 mths. later original lesions had extended.
12.	I.M.18	not known	L. scapular area	Paint and Pills b.d.	5 wks.	Hanovia 18″ 5—25 mins.	2nd degree erythema	Islands	Repigmented steadily with small islands. When last seen progressing well. Not seen after intermediate stage; patient disappeared.
13.	I.F.13	not known	Forearms and temples	Paint only		sun 20 mins.	4th degree erythema	Islands	Steady repigmentation.
14.	I.M. 14	not known	Periorbital region and ant. hair margin	Paint only		sun 5 mins.	4th degree erythema	Islands	Repigmented.

TABLE I (CONTIN.)

Case No.	Race Age and Sex	Duration	Areas Affected	Method of Treatment	Duration of Treatment	Method of Irradiation	Reaction	Signs of Repigmentation	Remarks
15.	I.M.42	1 mth.	Angles of mouth	Paint Paint and Pills	10 wks. after 6 wks.	T.W. contact 5—10 mins.	4th degree with vesiculation		Some encroachment of pigment from periphery. 50% repigmentation.
16.	I.F.13	not known	L. ear area L. elbow both legs	Paint and Pills b.d.	10 wks.	Hanovia 18" 5 mins.			Complete repigmentation of all areas except legs which when last seen were showing islands of pigment.
17.	I.M.32	5 yrs.	Forearms, hands, chin, forehead, between shoulder blades, legs	Paint and Pills j.b.d.		Kromayer contact 35 mins.			Beginning to repigment after 4 mths. intensive treatment.
18.	A.M.24	1 mth.	Patchy on hands	Paint and Pills j.b.d.	2 mths.	Hanovia 10" 10—20 mins.	1st degree erythema		No sign of repigmentation.
19.	I.F.17	1 wk.	Back of neck. L. ankle	Paint only	6 wks.	sun 20 mins.			No reaction so far but has only had two treatments.
20.	I.F.14	not known	L. knee behind R ear	Paint only	5 wks.	Hanovia 10" 10—20 mins.			Absented after promising initial response.

TABLE II. ANALYSIS OF RESULTS

Distribution	No. of Cases	No. Repigmented	Failures	Doubtful
Face, head, neck	14	6 (43%)	6 (43%)	2 (14%)
Trunk	7	3 (43%)	4 (57%)	—
Arms	6	4 (66%)	1 (17%)	1 (17%)
Legs	7	3 (43%)	1 (14%)	3 (43%)
Total	34	16 (47%)	12 (35%)	6 (18%)

of a person of average pigmentation. The duration of exposure required to produce this erythema, at a given distance of the source from the skin, is regarded as the 'minimal erythemal dose'. This is determined for every lamp and serves as a standard by which ultra-violet dosage may be estimated and prescribed.

In the present series of cases all but one were darkly pigmented, making the detection of a true erythema not impossible but of unreliable accuracy. Further the position was complicated by the common tendency of this series of cases, when exposed to sunlight, to exhibit the so-called Meirowsky phenomenon,[9] i.e., the skin fairly rapidly undergoes a process of primary pigmentation or pigment darkening. Kooij and Scott,[10] working recently on primary pigmentation of normal skin in the African, along similar lines to the earlier work on Europeans done by Henschke and Schulze [11] and others [12] have found that the long ultra-violet wave-lengths of sunlight (2,900—3,900 Angstrom units) did not produce in their cases that erythema which precedes secondary pigmentation and is produced by the short wave-lengths (1,800—2,900). This being so, attempts to standardize the ultra-violet dosage in terms of time or wave-length appeared futile since exposed areas of vitiligo, normally unaffected by sunlight, when painted with Meladinin and exposed to the sun, developed in some cases a 4th-degree erythemal reaction after only 10 minutes exposure; whilst in other cases, or even in other areas on the same case, no visible reaction occurred after as much as 30 minutes contact-exposure to an efficient artificial short-wave ultra-violet source, emitting a known 2,537 Angstrom-unit wave-length. This variability of reaction to ultra-violet led to a cautious process of graded trial with each case and of each area to be treated.

The initial routine adopted in this series, particularly with areas which would be expected to react briskly to irradiation, was to apply the paint and advise the patient to expose himself to the midday sun for 15—20 minutes; if there was no appreciable reaction to this the painting was repeated in 2—3 days using U.V.R. from a Kromayer, a Westinghouse thin window, or a Hanovia lamp. Where possible treatment was given thrice weekly and, if there was little reaction, pills containing 10 mg. of ammoidin and 5 mg. of ammidin were also given in dosage of 1 or 2 daily. An exception was one African (case 18) who received the pills t.d.s., was painted thrice weekly, and received U.V.R. from a Hanovia lamp at 10 inches up to 30 minutes; the only reaction to this was an erythema with no sign of repigmentation.

The degree of repigmentation not only varied from patient to patient but from lesion to lesion and even in different parts of a lesion. One interesting anomaly was observed in case 2. When first treated he was given paint alone on the abdominal lesion followed by U.V.R. to that area; within 3 weeks he developed an acute

Fig. 1. Case 1 before treatment.

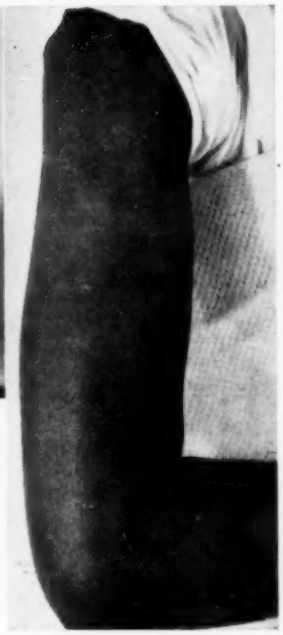

Fig. 2. Case 1 after 8 weeks' treatment.

Fig. 3. Case 6 before treatment. *Fig.* 4. Case 6 after 2 weeks' treatment.

vesicular eruption over the leucodermic areas on the shins in spite of the fact that these had received no treatment in any form and were protected from sunlight by long trousers. Repigmentation developed in these areas in the usual 'island' way but was not complete, leaving some depigmented areas. There was no repigmentation in his forehead in spite of intensive treatment there. The lesion on the abdomen after receiving 30 minutes of 'thin window' U.V.R. treatment (at contact distance instead of the normal 6 inches) eventually developed an acute vesicular eruption which led to the development of islands of pigment within 24 hours; repigmentation then continued in the usual way. Case 9 showed complete repigmentation of a lesion on the back, but no response on the legs.

Case 11 started to respond well but then ceased to come for treatment, and when the patient was seen 4 months later the previous lesions had extended although the repigmented areas had not relapsed. Further treatment was producing good results when the patient again disappeared and has not been seen again. Case 5 showed the most dramatic response of all and repigmented completely after 1 treatment. Case 6 also repigmented very quickly, giving an excellent result in less than 2 weeks. Case 12 was reacting very well to treatment and had reached the 'island stage' when treatment had to be stopped for 6 months while the patient retired to prison; he is therefore labelled as 'doubtful' in the assessment of figures.

It will be seen, therefore, that some of the cases described as failures have hardly given the treatment a fair trial, while two or three of the more recently treated cases have not been under observation long enough to be regarded as outright failures although they may show as yet no signs of repigmentation. The analysis therefore in Table II, is a very conservative one and probably underestimates the effectiveness of the treatment. One significant observation was the complete failure to repigment in the African cases in spite of the most intensive and protracted treatment. The one European case was also a failure; although she stopped treatment after 2—3 weeks, there was very little suggestion of repigmentation even after an acute vesicular response.

As already observed, an acute vesiculation was obtained with 40% of the cases, which led to some modification in the treatment. After surveying the results the general impression was that the best results were obtained in cases in which an acute near-vesiculation reaction was deliberately provoked and the maximum safe dosage was given as early in the course as possible before the skin had time to acclimatize itself to the photodynamic effect of the drug.

The method of repigmentation was in most cases by the development of minute perifollicular islands of pigment which gradually coalesced; this phenomenon is well shown in the photographs. The other method of repigmentation is that of peripheral encroachment which was well illustrated in case 15, where the lesions round the angles of the mouth materially decreased in size but where no small islands developed.

One or two cases did develop larger 'freckle-like' lesions which came to nothing and disappeared as soon as the U.V.R. was stopped. This was particularly noticeable in case 9 on the legs, which failed to repigment, and in case 2 on the forehead.

As a result of these observations it is planned in future to use more intensive treatment initially, trying to avoid a reaction of such severity that it interferes with regular treatment. Before it is possible to estimate the optimum dosage for each case and lesion, considerable experience is required; from the present results it is suggested that,

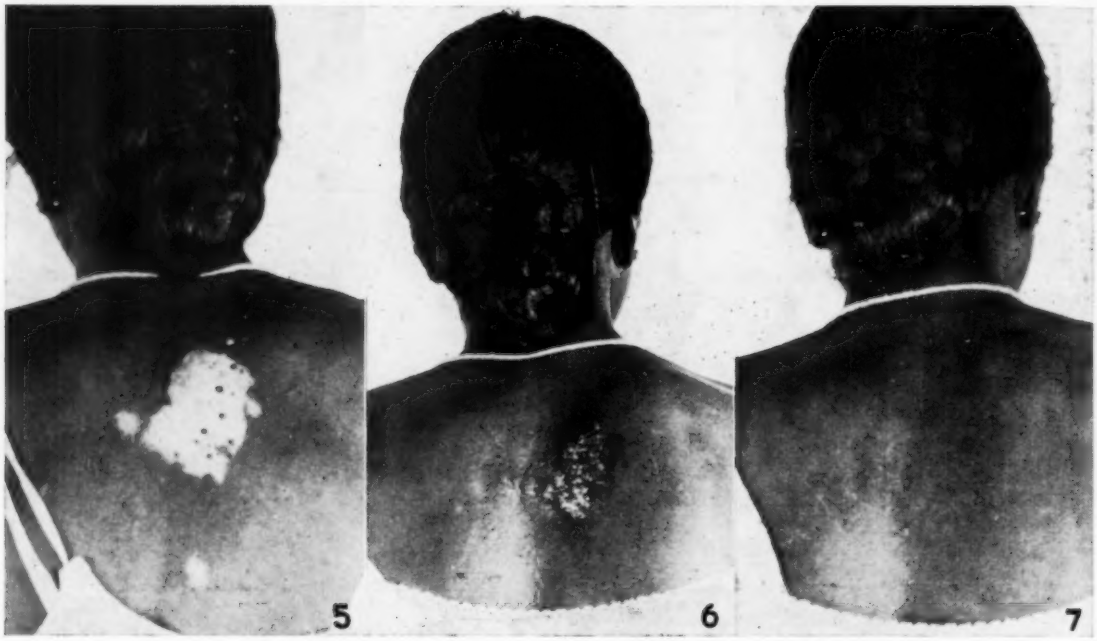

Fig. 5. Case 9 before treatment. *Fig.* 6. Case 9 after 6 weeks' treatment. *Fig.* 7. Case 9 after 14 weeks' treatment.

unless there is some definite sign of repigmentation following vesiculation within 2 weeks, a satisfactory result is unlikely to follow. It also appeared that better results were obtained when both paint and pills were given from the beginning, in addition to the maximum dosage of U.V.R. which the skin could tolerate.

It is not possible as yet to hazard more than a guess as to the mode of action of this drug. It is possible that it inhibits the action of certain substances containing the sulph-hydryl group which antagonize the enzymatic action of tyrosinase in the normal formation of melanin, but as yet there is no supporting evidence for this.

CONCLUSION AND COMMENTS

In summing up our results and trying to give rough statistical figures, the lesions are divided into 4 groups. The reason for doing this is that it is extremely difficult, as already explained, to regard a patient with several lesions as cured, because while some lesions have repigmented others may show no change; the extreme example was shown in case 2 where repigmentation took place in the greater part of the leg lesions, but an area of depigmentation remained which in spite of extensive treatment would not repigment. In Table II cases have been classified according as the vitiligo affected (i) the head and neck (14), (ii) the trunk (7), (iii) the arms (7), and (iv) the legs (8).

The only justifiable comment to be made on these results is that this drug offers a means of treating vitiligo with a reasonable prospect of success. One observation made in these cases was that the soft satin-like type of skin appeared to react very well while the coarse hyperkeratotic areas or type of skin was less likely to repigment. Attention is drawn, however, to certain discrepancies between these results and those of the original Egyptian and French workers. In this series a brisk inflammatory reaction in the skin has been almost a necessity to produce repigmentation. This observation, so contrary to those of Egyptian and French workers might be partly due to the well-known tolerance of pigmented skins to U.V.R. It has also been found that smaller and more recent lesions have reacted best and it would appear that if patients could be treated early on in the course of the disease before lesions had become extensive, results would improve considerably.

The method is rather laborious and it is felt that it should only be carried out under regular supervision and with carefully regulated U.V.R. exposures. It is obviously more convenient, and safer, to treat such cases in a special hospital department under the supervision of a trained physiotherapist. Casual and occasional treatment with the drug, followed by haphazard irradiation, is very strongly deprecated and the result in case II, with its extension of lesions, supports this view. There is also some possibility that the drug can provoke a permanent light sensitization. Sidi and Bourgeois-Gavardin [8] in Paris report 5 such cases in a series of 106 cases, and in this country of sunshine that is hardly a desirable condition to bring about.

This paper is in the nature of a preliminary report, but the results would seem to justify further and more complete trials of this treatment.

We should like to thank Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, Durban, for permission to publish these cases, and also Miss Campbell, Mrs. Hennessy, Mrs. Pearse and Mr. Dunlop, the physiotherapists whose willing co-operation was most helpful in carrying out this work. We also wish to thank Miss McLaggan, Clinical Photographer to the Province, for the photographs taken at various stages.

SUMMARY

A report on the result of treatment of 20 cases of vitiligo with Meladinin is presented. Two long-standing African cases failed to respond and treatment was unsuccessful in the one European case. The remaining Indian cases responded well to treatment, showing repigmentation in more than 50% of the lesions.

POSTSCRIPT

Since this paper was written further work has been carried out by the authors along these lines particularly with European cases. It has been found that white skins show a much more marked photosensitization in areas where Meladinin paint has been applied than do Indian or African skins. As a result of this it is strongly recommended that in treating such cases an initial application of a 25% solution of the paint in S.V.R. be used and exposure of treated area be limited at first to a maximum period of 5 minutes, the strength of the paint and the duration of exposure can then be gradually increased.

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GASTRIC DIVERTICULA

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Gastric diverticulum is a comparatively rare condition. It was first described by Moebius in 1661 and later by Fourier in 1774.[5] Radiologically it was first demonstrated by Brown in 1916. According to Feldman [3] the radiological incidence is 0·4% and the autopsy incidence 0·2%. Statistics available (1946) indicate that about 155 cases have been reported in the literature.[7] This apparently innocuous pouch, whether congenital or acquired, is not infrequently responsible for disturbed health.

Schmidt and Walters' classification,[8] which is based on the commonly accepted aetiological factors, recognizes:

1. Congenital ('true') diverticulum—in which all layers of the stomach are intact. These are due to malformation or interrupted development during foetal life.
2. Acquired ('false') diverticulum, of which there are 2 types:

(*a*) Pulsion diverticula, which are due to increased pressure and are mucosal hernias through weak spots in the muscular wall.

(*b*) Traction diverticula, resulting from perigastric adhesions due to inflammatory conditions.

The commonest site is at the cardia. Feldman [3] states that 85% occur at the cardia on the lesser curve (on the posterior wall just below the cardio-oesophageal opening). Other sites are rare, though cases have been described at the pylorus and the greater curvature.

The aetiology is difficult to attribute to any particular cause. Tracey [9] points out that 'an anatomical weakness exists near the cardiac orifice, due to a thinning of the circular and oblique fibres at that site, while the longitudinal bundles continuing down from the oesophagus divide into two muscular groups at the cardia'. This localized point is therefore comparable to that existing at Killian's dehiscence, where oesophageal diverticula occur. Review of the literature shows that diverticula are found most frequently in middle life. This may suggest that the general loss of muscular tone in middle age may be another contributory factor in the causation of pulsion diverticula. The fact that the cardia is a pressure point for all food entering the stomach and is also the site for the passage of large blood vessels and ducts through the wall are other points worth mentioning. A curious fact is that gastric diverticula are a common occurrence in monkeys and hogs [1] and some writers suggest that in the human this may be a form of atavism. The study of these cases has been prompted by certain unusual features which they exhibit, the association of clinical symptoms with this generally assumed harmless condition, and the difficulties and pitfalls in the radiological diagnosis.

CASE REPORTS

Case 1. M.J.E., a European woman aged 62, was admitted to hospital on 10 September 1951 complaining of epigastric discomfort, pain over the left costal margin, distension and very severe belching, of about 4 months' duration. Food aggravated the pain, while eructations and lying down relieved it. Her appetite was poor and she was afraid to eat because food seemed to 'stick' at the epigastrium. She had lost about 16 lb. in weight. There was no history of haematemesis, melaena or vomiting. She had a similar episode 2 years ago, when she received conservative treatment for dyspepsia.

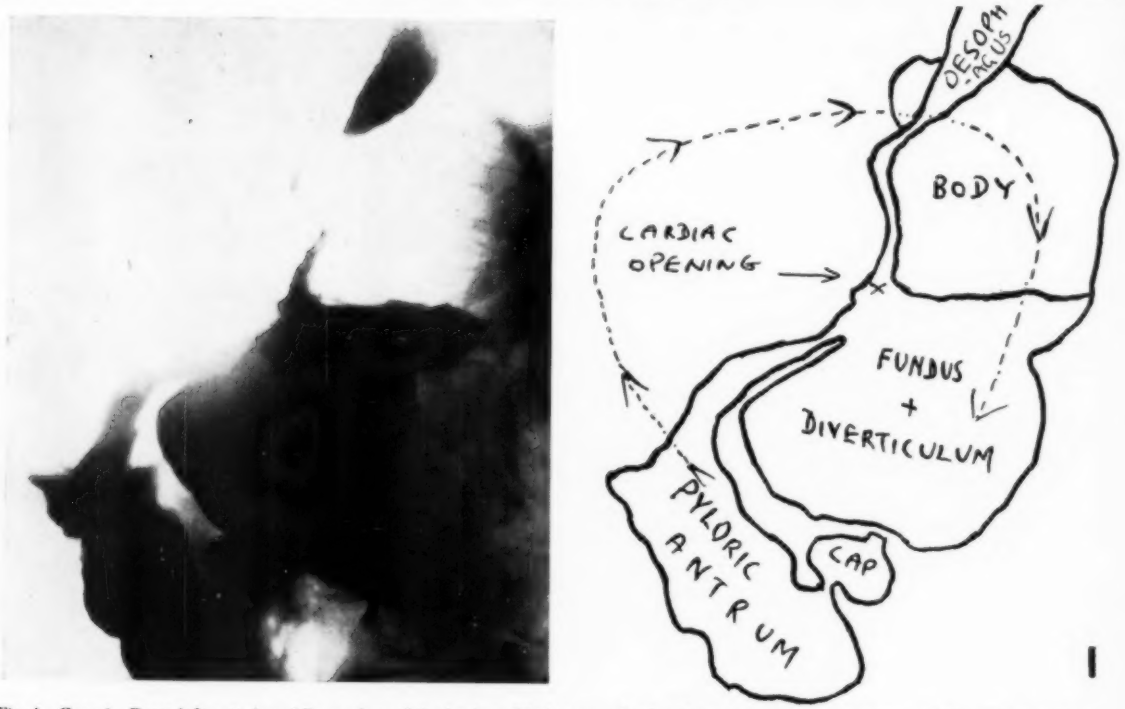

Fig. 1. Case 1. Erect left anterior oblique view of the barium-filled stomach showing the gastric volvulus. Note pyloric antrum points downward as is usual in this type of volvulus. See accompanying diagram. Arrow indicates direction of rotation of the stomach which resulted in a volvulus.

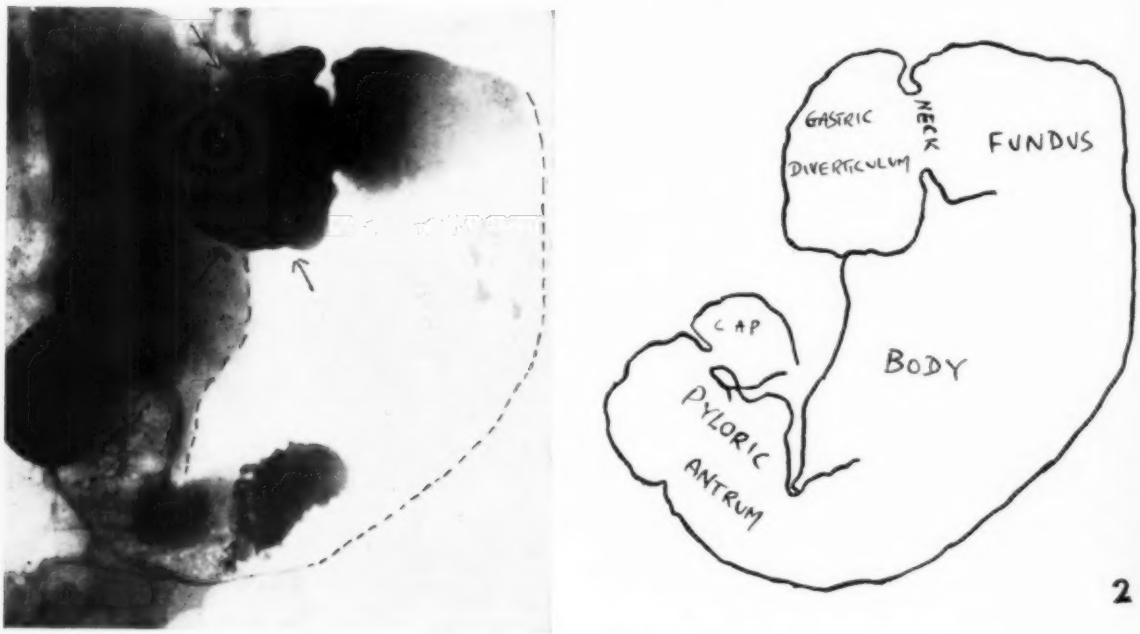

Fig. 2. Case 1. Supine right anterior oblique view showing the large gastric diverticulum with a wide neck. See accompanying diagram.

There was nothing else relevant in the present or past history.

On examination the only objective clinical signs were constant belching accompanied by 'weird noises' and aggravated by deep breathing. There was also a constant passage of flatus. No succussion splash was heard over the stomach and there was slight tenderness in the left hypochondrium.

The blood picture was normal. The blood urea was 28 mg. per 100 ml. Serum protein: albumin 5·1 g. per 100 ml., globulin 1·6 g. per 100 ml. The electrocardiogram was normal. The cholecystogram made in the out-patient department was normal.

A barium-meal examination made 3 years prior to admission showed 'a biloculated stomach of the 'cup and spill' type. No other abnormality seen.' A similar examination made 2 years prior to admission showed 'marked gaseous distension of the stomach' and small and large bowel. The stomach presented the appearance of a partial volvulus. No other pathology was noted and no cause for the volvulus was discovered'. Barium studies following the present admission revealed: 'A volvulus of the stomach on the cardiopyloric axis of the organo-axial type. The greater curvature had swept upwards and underneath the diaphragm (Fig. 1). The oesophageal entrance is now on the lateral side of the stomach. This volvulus is associated with a large gastric diverticulum of the cardia (Fig. 2). The follow-through also showed diverticulosis of the colon. Stasis in the diverticulum was present 6 hours after the examination (Fig. 3)'.

Surgical exploration disclosed a capacious stomach with a bulge medial to the spleen. The spleen itself was extremely mobile and it was suspected that it might be partly responsible in the production of the volvulus by traction on the stomach. A splenectomy was performed. A distinct gastric diverticulum was then brought into

Fig. 3. Case 1. Prone 6-hour film showing stasis in the large gastric diverticulum.

Fig. 4. Case 2. Erect postero-anterior view of stomach showing (1) the irregularity at the fundus resembling an ulcer.

view. Its neck was 2 inches in diameter and the body 3½ inches across. A diverticulectomy was then performed. The rest of the stomach and bowel appeared normal.

Symptoms recurred 6 months later and the partial volvulus was still noted on radiological examination. A partial gastrectomy was performed subsequently. At present, 2½ years after the operation the patient is well.

Case 2. E.J., a European male aged 63, was admitted to hospital for stabilization of his diabetic state, from which he had suffered for the past 25 years. Apart from his diabetes he gave a 2 months' history of distension and the passage of large amounts of foul-smelling flatus by the mouth. He had lost about 15 lb. in weight. There was nothing else of importance in his present or past history.

The physical examination was essentially negative except for bilateral early cataracts. A provisional diagnosis was made of diabetes mellitus with query carcinoma of the stomach. A barium-meal examination performed 2 months before admission had revealed no abnormality.

On the 2nd day after admission, 3 haematemeses of 'coffee grounds' vomit took place, totalling 2 pints. The blood pressure dropped to 85/65 mm. Hg and ketosis developed. Whole blood, 500 c.c., was immediately given and the appropriate treatment for ketosis instituted. Recovery and stabilization was reached 48 hours later.

A week after this recovery from haematemesis a barium-meal examination was performed. 'Some irregularity was noted in the region of the fundus of the stomach, the nature of which was indeterminate, and re-examination was suggested to exclude malignancy. The rest of the stomach and duodenum were normal.'

Ten days later the barium meal was repeated and again the deformity in the fundus was seen. 'There was a persistent fleck of barium at the fundus high up, with rugae radiating towards it. The appearances were strongly suggestive of a healed or healing ulcer.' A barium enema, a cholecystogram and a radiograph of the chest all appeared to be normal.

The patient was discharged and returned periodically as an out-patient. His diabetes remained well controlled, and he gained 8 lb. in weight in the course of the next few months. Clinically he felt reasonably well, but still complained of distension, especially towards the evening and when lying down, epigastric discomfort, and flatulence with foul-smelling eructations.

Three months after being discharged he presented himself for

Fig. 5. Case 2. Supine left anterior oblique view of stomach showing (1) pear-shaped smooth diverticulum, (2) narrow neck, (3) level of cardio-oesophageal opening.

another barium-meal examination. The irregularity and convergence of rugae at the fundus simulating a healed ulcer were noted again (Fig. 4). When the patient was placed in the supine position, a pouch appeared near the upper pole of the fundus; this was localized to the posterior wall near the greater curvature, at a level above the cardio-oesophageal opening (Fig. 5). The diagnosis of a gastric diverticulum then became obvious; the films taken show a well-defined, smooth, pear-shaped projection with a narrow neck. Barium remained in the diverticulum for over 6 hours (Fig. 6). Tenderness was not elicited, but the area was inaccessible to palpation. There was no spasm near or opposite the diverticulum and the mucosal pattern of the fundus was otherwise normal. The rest of the stomach and duodenum showed no abnormality.

DISCUSSION

The prevailing general impression is that gastric diverticula are seldom associated with symptoms. Love [4] mentions that this condition receives much less recognition in England than in the United States. Moses [6] states that 1 in every 3 diverticula will give rise to adverse symptoms. According to Fawcitt [2] 'if evidence of an ulcer, neoplasm or gall-stones cannot be found in a patient who complains of unorthodox dyspepsia, flatulence, pain in the chest, etc., and who is not suffering from an obvious condition, then the presence of diverticulum may be suspected'. The symptoms which the diverticulum of a stomach may cause are not specific. They are many and varied. Stasis and decomposition of food may cause irritation, inflammation and ulceration.

Both the cases reported above exhibited symptoms which appear to have been due to the gastric diverticulum or the complications which it has precipitated, viz. a gastric volvulus in case 1, and haematemeses in case 2, with subsequent failure of diabetic control.

Case 1 is a rare and interesting one. A large gastric diverticulum appears to have been the primary cause for the gastric volvulus, though the extremely mobile spleen was possibly also a contributory factor. No similar record of a gastric diverticulum associated with a gastric volvulus could be found in the available literature.

An interesting feature about case 2 is the relatively uncommon site of the diverticulum, being on the greater curvature, near the upper pole of the fundus at a level above the gastro-oesophageal opening.

Gastric diverticula may be easily missed on radiological examination. The first 2 barium studies in case 1, and the first 3 in case 2 failed to reveal the condition. Feldman [3] states, 'Many cases are overlooked in routine radiological examination'. Our case 2 would suggest that this may occur particularly in the presence of a narrow neck (Fig. 5). Such a situation may give rise to a serious diagnostic problem. In all cases of unexplained symptoms referable to the upper gastro-intestinal tract, and in which barium studies are negative, the possibility of a gastric diverticulum should be investigated. A slight modification of the routine technique is suggested. This involves the examination of the patient in the supine and prone position soon after the first few mouthfuls of barium have been swallowed, for otherwise a full fundus may obscure the diverticulum. Time should be allowed for the diverticulum to fill, because a narrow neck may be present. It is therefore advisable to re-examine the patient after he has been lying on his back for 10-15 minutes.

SUMMARY

1. A rare case of a gastric diverticulum associated with and causing a gastric volvulus is recorded.

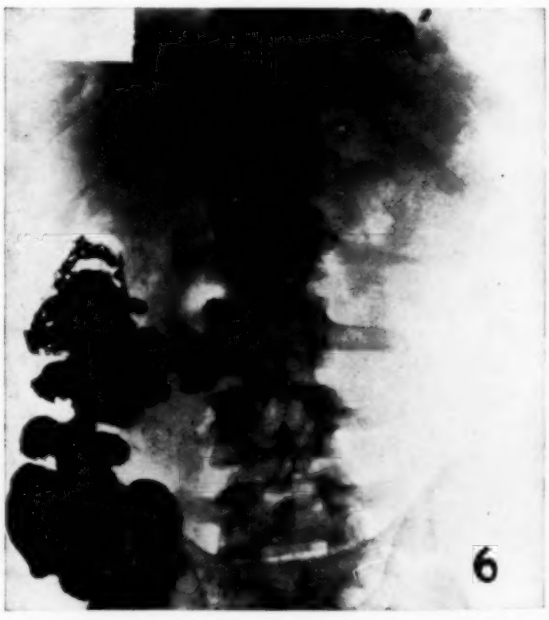

Fig. 6. Case 2. 6-hour film showing retention of barium in the diverticulum.

2. Another case of a gastric diverticulum at an uncommon site and associated with symptoms is recorded.

3. The literature is briefly reviewed with reference to the incidence, classification, site, aetiology and symptomatology.

4. The difficulties which may be encountered in the radiological diagnosis are discussed.

I wish to thank Dr. Josse Kaye, Chief Radiologist, Johannesburg General Hospital, Mr. A. Lee McGregor, and Dr. Julius Buch for their advice and permission to publish the cases. My thanks are also due to Mr. Broadway for the reproductions of the radiographs.

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REVISION SERIES

VII. THE NERVOUS BREAKDOWN

AN APPROACH FOR GENERAL PRACTITIONERS

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'And the Lord God formed man of the dust of the ground, and breathed into his nostrils the breath of life; and man became a living soul.' (Gen. II, 7)

'Therefore, good Brutus, be prepared to hear:
And, since you know you cannot see yourself
As well as by reflection, I, your glass,
Will modestly discover to yourself
That of yourself which you yet know not of.'
(Julius Caesar, I, 2)

Considering our present stage of scientific development it is anomalous that a group of diseases should be designated by such a non-descriptive term as 'nervous breakdown', particularly when sufferers from nervous disease are variously estimated to comprise 10-20% of all illness, and to complicate about 30% of the remainder. A psychoneurosis or neurosis is an illness with specific etiology, pathology, symptomatology and treatment. For various reasons the natural history of the neuroses have as yet not been clearly defined. A vast pool of empiric knowledge exists about them and many theories of pathology, or more precisely psychopathology, have been evolved. These have permitted a clearer understanding of the diseases and a more rational management; it is unfortunate, therefore, that in the evolution of these concepts terminology was employed which tends to obscure existing knowledge.

A psychoneurosis is an illness of which the symptoms are an effort to re-establish a satisfactory equilibrium between the individual and his environment. This state of equilibrium is not static; the individual is constantly readjusting so that he remains within an optimal range, a process which may be likened to the heat-regulating mechanism of the body. Physiologically the heat-state equilibrium can be measured and a normal optimal range for the individual has been charted. When the individual becomes pyrexial, i.e. the range of heat equilibrium extends beyond the normal, the individual (though unaware of any norms) is able to appreciate that he is ill. And similarly, when behavioural patterns cease to remain within an optimal range of equilibrium, again the individual knows he is ill.

Recently Burt [1] defined personality as 'the whole system of relatively permanent tendencies which are distinctive of a given individual and determine that individual's adjustments to the material and social environment.' It is when these 'adjustments' prove unsatisfactory to a degree demanding medical attention that the individual is mentally ill, i.e. he is said to have had a nervous breakdown. Implicit in this definition of personality is the concept of a *unique* individual with innate and acquired tendencies inter-acting with his environment, which is primarily psychological or social.

Nosological designations for various psychoneurotic syndromes have been in common usage for many years. Because of a lack of precise knowledge of etiology and pathology, they are ill-defined; such names as anxiety state, reactive depression, neurasthenia, obsessional states, hysteria, etc., have been and are in common use. Many are satisfied with the use of the term 'neurotic' or 'hysteria' as a designation of all psychological illness: vaguely, a neurosis is thought of as a junior psychosis and a psychosis as a state where the patient is really mad. The exasperation exhibited towards the psychoneuroses (which stems from a lack of knowledge about them) leads to an attitude which regards the symptomatology as purely imaginary on the part of the patient; if he wanted to, and had more 'guts', he would 'snap out of it'. There is a feeling of something reprehensible about this type of illness.

The diagnosis of psychoneurosis is frequently made only after exhaustive examinations and investigations, i.e. by a process of elimination.

Because of the above attitudes and beliefs which I

think are common, and which I, as a general practitioner held, I think it desirable to make several categorical statements.

DEFINITION

Where the individual's behaviour patterns are abnormal but where their objective is the re-establishment of equilibrium within an optimal range, the individual is suffering from a psychoneurosis. Where the behaviour pattern bears no apparent relationship to the re-establishment of equilibrium, a state of psychosis exists. In these conditions the patient's symptoms (behaviour patterns) are not imaginary, nor are they under control of the patient's will. He is as distressed, and the symptoms are as real, as those of the organically ill person; and like the latter, he is unable to 'snap out of it.' For this reason, the illness merits the same respect and attention as one caused by disease of an organ.

A positive diagnosis must be arrived at by an intelligent appraisal of the clinical features. This is stressed by Paul Wood[2] in his Goulstonian lectures: 'Medical officers . . . . must learn to diagnose neurosis on positive grounds: no greater blame can be attached to a psychiatrist who fails to make a physical examination than to a physician who fails to probe the mind. A few pertinent questions and the ability to listen to the replies are all that is required; lengthy details are not needed for diagnosis.' On this statement the *British Medical Journal* observed:[3] 'The place to prevent psychological casualties is in the front line, and the methods are those of *general practice rather than the hospital ward*' (the italics are mine).

Psychological illnesses present in varying degrees of complexity and intensity, and accordingly varying methods of treatment are necessary; the general practitioner is able to deal adequately with most cases he encounters, though some will require reference to a psychiatrist.

In order to deal with the psychoneuroses it is necessary to have (1) an awareness of the condition, (2) some knowledge of the disease, and (3) a discipline with regard to history, examination and treatment. These factors will now be dealt with:

THE CLINICAL APPROACH TO PSYCHONEUROSIS

1. *An Awareness of the Condition.* This implies the regarding of the individual as a *unique dynamic personality*, one constantly interacting with his environment and who brings genetic and learned behaviour patterns to each situation. His behaviour has meaning in terms of these forces. The basic patterns of behaviour are learned early in life and, with slight modifications, are those used to engage the stresses of life as they appear in the evolution of the individual. They are like the handwriting, the basic pattern of which one learns in childhood, and which one modifies as the years go by.

This awareness implies that the approach to the patient's complaint will take into consideration psychogenic as well as organic factors.

2. *Some Knowledge of the Disease as a Clinical Entity.* In reviewing my case histories from general practice, I found groups of symptoms presented by patients. In these cases I ultimately arrived at a diagnosis of psychoneurosis only after exhaustive examination and investigation. These symptoms rarely occurred singly—they usually consisted of several of the following:

Feeling of lassitude, tiredness, no energy.
Sleep disturbances—always sleepy, inability to fall asleep, waking after a short period of sleep.
Sighing and breathing disturbances—asthma, inability to take a deep breath.
Headache—usually on vertex; very common.
Gastro-intestinal disturbances—lump in throat, abdominal pain, constipation, diarrhoea, anorexia, wind.
Emotional disturbances—feeling pent up, nervous, anxious, worried, afraid, irritable, depressed.
Pain—precordial, in limbs, back.
Heart—palpitations, heavy feeling, heart conscious.
Dizziness, tremor.
Genito-urinary—impotence, premature ejaculation, vaginismus, dyspareunia, frequency of micturition.

Case 1. A 44-year-old female, complaining of feeling tired; periods longer than usual; severe palpitations particularly at night; choking feeling on walking fast; headaches; deep sighs and heart conscious. Duration: 3 months.

Case 2. A 23-year-old female, complaining of shakiness, lack of energy; depression, tightness around the throat, palpitations and domestic unhappiness. Duration: 3 months (since birth of child). Diagnosed as an 'anxiety state'. The importance of the depression was not appreciated until a suicidal attempt was made.

These case-histories can be matched by any general practitioner from his own files. Neither case had an organic basis. Case 2 is valuable because it illustrates the importance of a depression immersed among the other complaints. Here the depression was thought to be a reaction to domestic unhappiness, but this case was in fact a psychosis; suicidal threats were not given the respect they merited—a common attitude. *It cannot be sufficiently stressed that a patient who threatens suicide will probably attempt it. He is as seriously ill as anyone with a disease where death is a probable end-result.*

Most psychoneuroses present a cluster of symptoms which, if taken together, indicate the probable psychological origin. Any single complaint alone may be a symptom of a non-psychogenic disease; whereas the picture formed by all the symptoms grouped together is unlikely to be produced by organic disease. This is illustrated by case 1, in which there was mitral stenosis, though none of the symptoms were referable to this cause; marital infidelity on the part of the husband and financial stresses existed at that time, and when these improved, so did the patient's clinical state.

While it is desirable to recognize nosological entities, it is important to be able to recognize a syndrome as having a psychogenic origin. The severity of the condition can be assessed by comparing the intensity of the illness with the magnitude of the provoking stressful features.

3. *Discipline with regard to History, Examination and Treatment.* A rational discipline implies some rational concept of the genesis of disease.

Basic behaviour patterns are established during the early phases of development. In considering their evolution, one should start from the moment of fertili-

zation, at the time of union of the sperm and ovum, for each brings with it its genetic heritage. The fertilized ovum exhibits an inherent drive which through growth and differentiation is directed at the full potential evolution of the individual. The ultimate individual cannot achieve more than this inherent potential; e.g., if the potential height of the individual is 5 feet 8 inches he will not evolve to be a 6-footer. The full potential of the growing organism can be realized only if its needs are adequately met. For example, adequate nutrition and oxygenation is necessary *in utero* for the development of the foetus into a healthy baby. Experimentally-induced anoxic states in pregnant mice are followed by congenital abnormalities in the litter. Rubella in the pregnant mother may be followed by congenital abnormalities in the infant.

At birth the needs of the organism change. It now requires air *via* the respiratory passages, food *via* the gastro-intestinal tract. It has emotional needs as well as bodily. Where the needs of the organism are not satisfied, then part of the energy of the inherent drive to development is deflected in compensating for the lack of satisfaction; e.g., where the need for vitamin D is not satisfied, part of the developmental drive is deflected to thickening the bones so that they can meet stresses more adequately. In this deflection of the drive by the non-satisfaction of a need, there is obstruction, and an obstructed drive builds up tension. The emotional accompaniment of this tension is termed anxiety and its basic method of expression is by restlessness and crying. In the adult anxiety is expressed by restlessness; crying has largely been culturally suppressed. The picture of the restless, crying, ill-fed baby becoming placid and pleasant on correct feeding is familiar to all. One need of the infant is the establishment of an emotional bond with one important person (usually the mother), and failure to satisfy this need in infancy can lead to cachexia and death. This is graphically illustrated in Bakwin and Bakwin's book.[4] The behaviour of the evolving child is so determined as to keep the swing of anxiety resulting from varying obstruction to its drive (because of denial of its needs) within an optimal range.

HISTORY AND EXAMINATION

Thus an adequate history must take into account:

A. *Previous Influencing Factors*

(i) *Family History and Constellation:* The genetic heritage is explored by enquiring into familial illness; intra-uterine influences by considering the maternal health and age during pregnancy; the patterns learned by considering the social status and cultural restraint of the family; and the state of 'happiness' and its goals provides the initial group-stresses encountered.

(ii) Development during infancy and pre-school years is evalued. It is during this period that basic behaviour patterns are established.

(iii) School, occupational and marital histories indicate the patterns used by the individual and his adequacy in these environments.

(iv) The health of the patient and previous handicaps imposed by ill-health are noted.

B. *The Premorbid Personality of the Patient*

According to our definition, the distinctive tendencies which determine his adjustments to his environment are considered. These are evalued by considering his effectiveness socially, domestically and at work. His values and habits are reviewed; characteristic traits and temperamental features are noted; his fantasy life is considered as an index to his objectives.

C. *History of Present Complaint(s)*

This is usually taken first. During this period the patient is best allowed to speak with little interruption, and it is here that a friendly, sympathetic relationship with the patient is established. Particular note must be taken of the circumstances of origin of the complaint as described by the patient.

D. *Examination*

(i) *Physical.* This must be complete and exhaustive. It should not be repeated during subsequent interviews, for this would be interpreted by the patient as indicative of uncertainty on the part of the examiner.

(ii) *Psychiatric.* Behaviour, talk (form and content), attention, association, mood, compulsive thoughts, orientation, ideas of reference, hallucinations, memory, intelligence, general knowledge, judgment and insight are each considered. Psychiatric text-books set out satisfactory methods for this: Curran and Guttman's is a useful short work for this purpose.[5]

(iii) *Investigations* which may be indicated as a result of the above.

TREATMENT

This implies the management and care of a patient or the combating of his disorder.

Every patient, whether psychologically or physically ill, should be approached with the awareness that treatment commences at the moment of introduction. The patient believes that he is organically ill; he is anxious and takes a critical attitude with regard to his doctor. A non-critical, sympathetic approach is essential; the patient must feel that he is being accepted by the therapist. This may be difficult for the doctor to convey, particularly because many patients assume a potentially hostile and rejecting attitude. The patient is a unique dynamic individual; acceptance is conveyed to him when he feels that the therapist recognizes him as such.

There are certain prerequisites for treatment, viz. (1) a sincere interest and desire to help the patient; (2) confidence on the part of the doctor that *he can help* the patient; (3) an orientation that the target is not 'cure' but the solution of an immediate problem; and (4) knowledge that the patient gains considerable relief from being able to 'objectify' his problems by pouring them out to a receptive non-critical ear. Listening is more important than questioning.

Treatment, as stated, commences from the moment of introduction to the patient. From this moment the above attitudes must be brought into operation. During the physical examination it is wise to remark casually on the normal findings as they are encountered, since fears concerning the integrity of the heart, blood pressure

and lungs can thereby be unobtrusively allayed. When the history and physical examination have been completed and an assessment of the patient's state arrived at, a provisional hypothesis of the causation of his symptoms are given to him. It is stressed that this hypothesis is purely provisional and that modifications will be made as subsequent facts emerge.

During the recital of his history the patient has probably reviewed his personality for the first time. The evolution of behaviour patterns is explained to him: they may be likened to dress in that they clothe his self. Writing is discussed as an example of behaviour patterns; this aids the concept of altering behaviour patterns and the time necessary for such a change to be learned. The therapist is described as a mirror in which the individual can view his patterns. Once the patient perceives the reflection he can strive to adjust them.

Whatever the patient's basic behaviour patterns may be—and their importance should not be minimized—the cause of his neurosis lies in the present. It is the result of a conflict between his wishes, strivings and desires on the one hand and restraints—cultural and social—on the other. The patient may exhibit varying degrees of awareness towards these conflicts.

Case 3. A 23-year-old married man complaining of frequency of micturition, anxiety, restlessness and tiredness. His main concern was frequency of micturition, which had commenced 4 months previously. He found it particularly embarrassing when in company or at work, and he was sure that he was being ridiculed by others because of his frequent visits to the toilet. At the time of the appearance of this symptom, his wife had suffered a miscarriage, in the sixth month of their marriage. While discussing this, he expressed the doubts that he had suffered as to his fertility because of a previous attack of orchitis following mumps. Reference was also made to masturbation, which he felt might have impaired fertility.

He was the only son of wealthy parents. He felt that he had been spoilt and somewhat mollycoddled as a youth, but in spite of this he was a virile athletic man, popular with his fellows. Recently he had negotiated a partnership, and he felt that he was too junior to occupy a position of the responsibility that it entailed. Since childhood he had been guided by his mother, and he still saw her daily and discussed his difficulties with her.

Before consulting a psychiatrist he had been treated by his general practitioner, by a urologist and by a physician. He had then become desperate about his condition, and been advised by his mother to see a psychiatrist. (In passing, it is worth noting how often the psychiatrist is visited on a note of desperation!)

Treatment: The initial interviews were devoted to the taking of the history and to the examination. During this period few questions were asked. The patient experienced much relief from anxiety during this narration. This allowed him to revert to parts of the history already covered and to supply information withheld in the first telling.

It was postulated that he was a dependent personality with a strong achievement drive; that in the presence of this dependent attitude, anxiety was engendered by his having to arrive at decisions demanded by his domestic and business environments. His fears with regard to sex and consequent infertility were related to his attitude towards his mother, previous masturbatory activity and the traumatic effect of the mumps orchitis.

The symptoms could be explained on the basis of being the somatic features of fear. This hypothesis was submitted to the patient, who was not able to accept or discard it. It formed the basis for subsequent interviews, where his history was examined in relation to this hypothesis. The genesis of behaviour patterns which emerged were then discussed. The factors leading to attitudes of dependence and feelings of inferiority and of not being accepted were brought to attention. No dogmatic attitude was adopted; the patient was allowed to come to his conclusions with little influence on my part.

At the end of each interview a summary of our discussion was given. He was seen for an hour once each week for 10 weeks. At the end of that period he felt relieved of his symptoms and was once more able to engage in his activities without discomfort.

CONCLUSION

Perhaps this article has not dealt with what is popularly conceived as a nervous breakdown, but the clinical picture envisaged here is commonly encountered; it is diagnosed positively and not by exclusion. It results from an inability of the individual to express his unique personality in his environment. An approach is described which takes this concept into account and which is calculated to aid satisfactory expression of the personality.

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THE INDICATIONS FOR CAESAREAN SECTION*

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Caesarean section is a dramatic operation which tends to bolster the ego of the surgeon. It is one of the easiest abdominal operations from the technical point of view, and therefore should not give rise to any sense of satisfaction. But unfortunately (or fortunately) there can be much basking in the reflected happiness brought to the mother and her family circle. Many evils are covered and sins forgiven by Caesarean section. The doctor doing obstetrics—as opposed to the one who has a special affinity for, and pride in, obstetrics—often resorts to Caesarean section in cases in which it conceals the inferiority of his obstetrical ability.

A critical analysis of this operation reveals that it is no less than an admission of failure; that is to say, failure on the part of nature in not allowing natural delivery or making it hazardous to the mother or child, or failure on the part of the medical attendant in not allowing nature to run its course. Inferiority of judgment,

* An address delivered to the Cape Western Branch of the Medical Association of South Africa on 24 September 1954.

or failure of perspective, or shortcoming in character, leads to a number of Caesarean sections. The operation is not time-consuming and it removes the anxiety of all concerned. To read that the incidence of Caesarean section has risen to 15% in certain areas tends to fill one with concern. There is no doubt that with modern methods of anaesthesia and resuscitative measures, and the aid of antibiotics, Caesarean section has indeed become a very safe procedure. Add safety to the simplicity of the operation and anyone can forecast an increase in its incidence. A safe increase is to be welcomed, not deplored; more mothers and babies are being saved. The great risk is an unnecessary increase in the incidence of the operation. In order not to lose sight of the *art of obstetrics* a knowledge of the broadened indications for Caesarean section is essential.

INDICATIONS FOR CAESARIAN SECTION

Cephalo-Pelvic Disproportion

As can well be understood, this abnormality should ever be the primary indication for Caesarean section. Every obstetrician the world over is agreed upon this score. However, it is justifiably felt that many sections are done for unproved disproportion. In a study of normal deliveries following upon an earlier delivery by Caesarean section Nel [1] (1954) found in an analysis of a series of cases at the Peninsula Maternity Hospital, Cape Town, and also of other series recorded in the literature, that most of these cases were normal deliveries of mothers who had been subjected to Caesarian section for supposed disproportion. To add insult to injury the babies born normally were almost invariably heavier than those previously delivered by the abdominal route. Faulty judgment is a false beacon that leads the unwary off the true course. A lack of confidence, and misplaced reliance on special investigations, are the usual evils unearthed when a thorough search is made—just as over-confidence may result in failure.

Should a patient with a questionable disproportion present herself, a careful clinical pelvic assessment should be made—under anaesthesia if required. X-ray pelvimetry, with its excellent rigid measurements, should, by good standards, be resorted to only after the true clinical picture has been obtained. No absolute reliance should be placed upon X-ray pelvimetry; the head still remains the best pelvimeter. The great unknown factors are uterine action and human reaction. The results of good uterine action are often astonishing. It must be kept in mind that our estimates of foetal size and skull flexibility are inferior. To judge the outcome of labour purely on mechanical measurements is therefore unduly harsh. Trouble presents itself immediately when uterine action falls short and the position of the foetal head is not favourable; and the skill of the obstetrician is immediately on severe trial. Much enjoyment can be derived from the skilful management of such cases. No one can lay down set times for the duration of a trial of labour. Too many variables are to be considered. The patient, her baby and her labour must be integrated and considered as one whole. Every case must therefore be studied and treated according to the mother's rights and the obstetrician's lights.

Placenta Praevia

Macafee's work [2] (1946) should always receive due credit, for in a way it revolutionized the treatment of placenta praevia. There have been many variations by other authors, but they have been of minor consequence. The basic principle established is that should the patient's bleeding have ceased the main objective is the survival of the foetus. In an institution the fully investigated patient is allowed to proceed with pregnancy until she is well over the 36- or 37-week mark, unless a complication intervenes.

The main complication, naturally, is a further haemorrhage; the other is the onset of premature labour. Should the patient start bleeding, treatment will depend upon its rate. A slow leak before viability requires careful watching, whereas a rapid one demands immediate intervention. Either complication—bleeding or labour—necessitates a vaginal examination in a fully prepared theatre. Whether Caesarean section is indicated depends upon the findings. Placenta praevia of types 3 or 4 usually requires Caesarean section, whereas the treatment of types 1 and 2 will depend upon the state and dilatation of the cervix, should the patient be in labour. It is emphasized that much can be learnt from abdominal palpation; e.g., whether the head can be pushed into the pelvis—easily said yet most difficult if not impossible to perform in many normal patients. Should the head be in one or other iliac fossa and return to the iliac fossa after manipulating it over the brim (in a patient with a story suggestive of placenta praevia), the strong suggestion is that the placenta is occupying the space on the opposite side. A high mobile head which overlaps the brim suggests a posteriorly situated placenta, whereas, should difficulty be experienced in palpating the head (i.e., as if something were between the head and the anterior abdominal wall), the suggestion is obvious.

Since viability and delivery by Caesarean section have become the basic principles in the treatment of placenta praevia both maternal and especially foetal survival rates, especially the latter, have improved markedly.

Incoordinate Uterine Action

This is a most distressing condition and one that leads to endless trouble for the busy practitioner. It exhausts not only doctor and patient but also the relations of both. Because of this all-round trial it is quite natural that an easy way out should be sought. On the other hand, should Caesarean section be decided upon after a prolonged unproductive labour, the question that invariably crops up is, 'Why was it not done sooner?' How these cases can be assessed is still utter mystery. The principles upon which decisions may be reached are the following:

1. *The Condition of the Patient.* By this is meant both her physical and mental condition. Should the patient be mentally negative to labour and practically 'without' herself I have no doubt that waiting only prolongs the agony and that no good can come from wasting valuable time. This statement is not one that allows for actual measurement, and therefore may be open to abuse. Should the patient's physical condition deteriorate despite adequate care there obviously is no sense in delaying the issue.

2. *The Condition of the Foetus.* Regular checking of

the foetal heart rate together with a careful study of the tone of the heart sounds is imperative. Signs of foetal distress, i.e. a rising foetal heart rate followed by abnormal slowing of the rate are usually the earliest signs of foetal distress. An irregular, slow foetal heart almost invariably denotes distress. It is obvious that a distressed baby cannot withstand the rigours of a long labour. However, it must be emphasized that the diagnosis of foetal distress is sometimes made when in actual fact there is no distress at all.

3. *Duration of Labour.* This is a vexing problem and one that has lead many obstetricians into laying down a time limit for labour in primary uterine inertia. Figures tend to show that should true labour last for longer than 48 hours the risk to foetal life becomes excessive. It must be taken into consideration that contraction and retraction affect the placental site and that interference takes place in the foetal blood-supply at irregularly recurring intervals in addition to a slight degree of permanent interference.

4. *Duration of Rupture of the Membranes.* Should the membranes rupture early in labour, as they are in the habit of doing in these cases, the contraction and retraction will have yet a greater effect on the foetus. In addition, the risk of infection becomes greater.

All these factors are to be gauged in conjunction with each other and together with the state and dilatation of the cervix, the nature of the contractions and the position of the foetal head. A Caesarean section cannot be considered in uterine inertia unless all or most of these factors are taken into account as one whole. It is the sum total that makes the medical attendant decide upon abdominal as opposed to vaginal delivery.

Constriction Ring Dystocia, should be considered in conjunction with incoordinate uterine action. Again it must be emphasized that it is only after proper assessment, and treatment aimed at relaxing the ring, that Caesarean section should be deemed the correct procedure.

Toxaemia of Pregnancy

Toxaemia of pregnancy on its own is a common indication for termination of pregnancy. Induction of labour by the calcium gluconate, hot bath, oil, enema, stripping and rupturing of membranes, followed by a pitocin drip if necessary, usually suffices. Caesarean section needs be resorted to:

I. Should the patient not respond to the induction of labour. Termination obviously is indicated and as the natural channels show no response the other way out may be chosen. Failure of response to induction has purposely been placed first as the acute variety can often be treated by sedation followed by induction of labour.

II. Should the toxaemia tend to become acute (in the fulminating variety the section should be done forthwith). The predominant symptoms and signs of the acute variety or, rather, the danger signs are: (1) rapid rise in blood pressure, (2) rapid increase in albumen, (3) oliguria or anuria, and (4) dimness of vision.

Foetal Distress

This is a most unsatisfactory indication for Caesarean section and is one which may allow no end of abuse. The diagnosis of foetal distress can only be based upon the following:

(*a*) A rising foetal heart rate.

(*b*) Abnormal slowing of the foetal heart rate during a contraction and failure to return to normal until near the end of uterine diastole.

(*c*) Irregular foetal heart beats.

(*d*) A loss of tone in the foetal heart sounds.

The causes of foetal distress early in labour other than the purely mechanical ones (e.g., compression of the cord) are most difficult to understand. It does fortunately not occur commonly.

Rarer Indications

Accidental Haemorrhage. This problem has not yet been settled, but there is some proof that accidental haemorrhage may well be the precursor of toxaemia of pregnancy and not follow as a result of it. O'Donel Browne [3] in 1952 drew forcible attention to the fact that Caesarean section in what he termed 'phase I' would indeed save the lives of innumerable babies. It is clear that a number of foetuses succumb after the warning haemorrhage. In cases in which the bleeding has been rather profuse, abdominal pain is present, and the foetal heart is heard, there is a case for immediate delivery. On the other hand it is well known that many patients give birth to normal babies normally after quite considerable antepartum bleeds (de Villiers [4]). The difficulty, as usual, is to know just where to draw the line. Once again each case has to be judged on its own merits. Very rarely, Caesarean section may be the only way out in severe cases of accidental haemorrhage, viz. in those cases in which the fundus rises, the girth increases in diameter, and the pulse deteriorates and/or the blood pressure drops, all in spite of adequate modern resuscitative measures.

The other single indications for Caesarean section can be listed as follows:

1. A 36-37 week pregnant *diabetic* not responding to induction of labour, or one in poor labour.

2. *Prolapse of a pulsating cord* through an os not fully dilated. It is important to keep pressure on the presenting part vaginally in order to prevent compression of the cord, until the section can be done.

3. Should a *vesico-vaginal fistula* have been operated upon successfully, or should a 'sling' operation for stress incontinence have been done.

4. *Pelvic tumours* blocking the passage of the baby are not common and should be carefully assessed before Caesarean section is embarked upon. Relatively large fibroids, anteriorly situated, are drawn out of the pelvis by labour. I have encountered a tumour on the posterior pelvic wall—proved to be a kidney subsequently—not giving the slightest trouble in the normal delivery of a normally sized infant.

5. Only very rarely is Caesarean section warranted for Rh incompatability. However, should a patient give a history of successive foetal losses due to this abnormality, Caesarean section, before term, at a time fixed so that all ancillary services (laboratory, paediatric and blood for replacement transfusion) can be at hand, does save babies.

COMBINATION OF CONDITIONS WHICH SINGLY DO NOT WARRANT CAESAREAN SECTION

Caesarean section may be very correctly indicated should a number of factors—none of which singly indicates section—present themselves in combination. These combinations are not uncommon in pregnancy and labour. In the presence of an abnormality, an additional factor often swings one into adopting operative procedures. It is re-emphasized that all these factors have to be weighed carefully in each case before the operation is undertaken. No absolute rules can be laid down. The late General Smuts's theory on holism applies very well here, viz. 'When all the factors are put together the "whole" outweighs the sum total'.

The following are a few examples of the combinations that may be found:

1. *Relative infertility* together with other abnormalities, e.g. toxaemia, abnormal presentation, incoordinate uterine action etc.

2. An elderly primigravida with the abnormalities listed above.

3. A patient suffering from *heart disease* or *tuberculosis* should not be allowed a trial labour and, therefore, should there be any doubtful disproportion Caesarean section is done.

4. In a *diabetic* with doubtful disproportion, a poor obstetrical history, or with no response or an inferior response to the induction of labour.

5. Signs of *pending uterine rupture* with an abnormal presentation.

6. *Toxaemia* with uterine inertia or doubtful disproportion, etc.

7. Post-maturity and foetal distress.

In these combinations a host of conditions may present themselves and the individual who has obstetrics at heart will weigh them and decide upon his mode of action. Should he decide against Caesarean section it must be remembered that this is an eminently reversible decision. Matters may just change—as is the nature of things—and a Caesarean become imperative.

During the 2 years 1952/1953, 648 Caesarean sections were performed in the maternity hospitals falling under the aegis of the University of Cape Town's department of Obstetrics and Gynaecology. In this time 14,151 mothers were delivered. Our hospital incidence of Caesarean section therefore is 4·58%.

The indications listed were as follow:

Disproportion	279
Placenta Praevia	78
Toxaemia	77
Incoordinate Uterine Action	52
Accidental Haemorrhage	21
Foetal Distress	10
Other Single Indications	55
Combination of Factors	76
Total	648
(Repeat Caesarean Sections	133)

A number under 'disproportion' should in actual fact come under 'combination of conditions' as occipito-posterior and inferior labour were added factors besides doubtful disproportion. True and doubtful disproportion oftimes are complicated by inferior labours.

CONCLUSION

At no time can hard and fast rules be laid down; human beings, and factors associated with them, are too variable. Every individual reacts differently to stimuli. A pregnant mother responds to the induction of labour, and to labour itself, in her own way. It is for the clinician to observe these ways, analyse them critically, and then keep an open mind upon his course of action. In addition it must be kept in mind that there exists what can almost be described as a maternal mental control of labour; that is to say, the type of labour often depends upon the patient's mental attitude towards it. Due attention should be paid to this concept antenatally; endeavours should be made to teach the patient self-reliance. On the other hand the patient's mental attitude often depends upon the type of labour she experiences.

The response to induction and the type of labour are all-important. Should control over these factors be attained the indications for Caesarean section and the incidence of the operation will drop markedly.

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NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

The Eli Lilly International Corporation announce the following:

'Ilotycin' (Erythromycin, Lilly), Otic, with Polymyxin B and Benzocaine. 5-c.c. size for Otic Solution.

Description: Each package includes as separate units: (1) One bottle containing 25 mg. 'Ilotycin' (as the glucoheptonate); (2) One bottle of diluent containing 50,000 units polymyxin B (as the sulphate), with benzocaine, 5%, in propylene glycol; (3) Dropper assembly. When the liquid portion is added to the 'Ilotycin' before dispensing, the resulting solution totals 5 c.c. in volume.

When mixed as directed, each c.c. will contain: 'Ilotycin' (as the glucoheptonate), 5 mg.; Polymyxin B (as the sulphate), 10,000 units; Benzocaine, 5%. After mixing, Otic 'Ilotycin' is stable for 30 days without refrigeration.

Indications: Since Otic 'Ilotycin' contains the most potent antibiotic against gram-positive bacterial infections ('Ilotycin') and the most potent antibiotic against gram-negative bacterial infections (polymyxin B), it is recommended for use in the great majority of infections of the external ear. This includes all three of the broad categories of external ear infections—the dry type with scaling and fissures; the wet, 'weeping' type; and furunculosis.

Dosage: Three or 4 drops should be instilled into the external auditory canal 3 or 4 times daily. As with any solution to be instilled into the ear, the patient's comfort is increased if the solution is warmed in the hand before use.

CANADIAN WEIGHT AND HEIGHT TABLES FOR WHITE SOUTH AFRICANS

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During 1953 a country-wide study was carried out in Canada by the staff of the Nutrition Division, Department of National Health and Welfare, on the heights and weights of Canadians in different parts of the country. The areas were statistically selected so that they were representative of the total Canadian population and within each area individuals were selected by random approaches to schools, homes and business establishments. The resulting figures are believed to represent the population with an accuracy of 5%. Some 22,000 people of all ages were measured.

As far as known to the authors this is the first application of country-wide sampling methods to a height-and-weight study. Other studies on height and weight (e.g. the survey carried out by le Riche[1] on Pretoria White school children) have not been based on a sample of the total population.

The Canadian data are presented in the hope that they will be found interesting and perhaps useful in South Africa.

The primary use of height-and-weight tables in paediatrics is for the study of growth rates in children. Amongst adults such tables are used mainly in relation to overweight and obesity.

The estimation of overweight is now being again reduced to relatively simple terms, using height and weight, and at times perhaps with reference to 3 different types of build. More complicated indices such as Pirquet's index, Brailsford Robertson's formulae and Tuxford's index (Paton and Findlay[2]) as well as the ACH index (Franzen,[3] Franzen and Palmer[4]) are seldom used. Stuart and Meredith's selected percentile tables for children[5] have been used more recently. The Wetzel grid[6] is a lineal descendent of Brailsford Robertson's growth curves. The grid is an interesting restatement of the observation that the growth curve of a healthy child is parallel to the mean curve of the social and racial group to which the particular child belongs.

While studies on body build and body type are interesting from genetic, metabolic and endocrinological points of view,[7, 8] such differences are not easily expressed in simple, measurable terms.

A different approach from that of body type is the use of newer ways of measuring body fatness or leanness in relation to height and weight. Skin-fold measurements were obtained in this Canadian study, and may be reported upon at some future time.

Whether obesity results from hereditary predisposition, or as is more often the case, from overeating, the selection of persons suffering from this condition, is most readily made by using an arbitrary statistical standard. We make the assumption, based on life-insurance experience,[9, 10] that heavy weights in adults, regardless of type and heredity, show an extra mortality. We suggest that for adults the mean weight at ages 25—29, with a variation of 10%, would probably be the most desirable weight. This suggestion is being tested for these Canadian averages by further statistical studies.

TABLE I. AVERAGE HEIGHT AND WEIGHT OF CANADIANS BY AGE AND SEX

(1953 Survey. Nutrition Division, Department of National Health and Welfare, Ottawa. May, 1954)

Age (years)	*Male*		*Female*	
	Average Height (inches)	*Average Weight (Pounds)*	*Average Height (inches)*	*Average Weight (pounds)*
2	34·7	30	33·6	28
3	36·6	32	36·0	31
4	39·2	37	39·2	36
5	41·9	40	41·8	41
6	44·6	46	44·2	44
7	47·0	50	46·5	49
8	49·1	57	48·9	57
9	51·3	63	51·0	62
10	53·5	70	53·3	69
11	55·4	77	55·3	77
12	57·4	84	58·2	92
13	59·3	94	60·4	102
14	62·2	108	61·3	107
15	64·7	119	62·2	112
16—17	66·7	136	62·5	120
18—19	68·0	144	62·6	124
20—24	67·9	154	62·8	124
25—29	68·3	160	62·7	126
30—34	68·0	167	62·8	130
35—44	67·5	167	62·4	135
45—54	66·9	164	61·8	144
55—64	66·0	161	61·3	147
65 and over ..	65·5	155	60·6	138

DISCUSSION

The well-known study by Dublin[11] on 200,000 insured white males shows that death rates for over-weight and obese males increase with weight increase, especially in regard to organic heart diseases, angina pectoris, arterio-sclerosis, acute and chronic nephritis, cerebral haemorrhage, cancer, diabetes, and to a lesser extent for accidents and suicides.

Whether these conditions are merely associated with obesity, or whether there are causative factors—as yet unknown, conjectural or partly known—which cause both obesity and these diseases are questions which cannot at present be answered. What we do know is that evidence is accumulating that dietary control can improve life expectancy for at least a certain number of these conditions.

For instance, experience in Leningrad during severe

TABLE II. CANADIAN AVERAGE WEIGHTS FOR HEIGHT AND AGE

(In ordinary indoor clothing, without shoes)

(Nutrition Division, Department of National Health and Welfare, Ottawa, Canada, June, 1954)

MEN

Height Ft.	ins.	15 yrs.	16—17 yrs.	18—19 yrs.	20—24 yrs.	25—29 yrs.	30—34 yrs.	35—44 yrs.	45—54 yrs.	55—64 yrs.	65 yrs. and over
4	11	92	99	116	121	128	134	135	127	138	126
5	0	97	103	119	124	132	138	139	132	141	130
	1	102	108	122	127	135	141	142	136	144	135
	2	106	113	125	131	139	145	146	141	148	140
	3	111	118	128	134	142	148	150	146	151	144
	4	116	122	131	138	146	152	153	150	154	149
	5	121	127	134	142	149	156	157	155	157	154
	6	125	132	138	145	153	159	161	160	160	158
	7	130	136	141	149	156	163	164	165	163	163
	8	135	141	144	152	160	166	168	169	166	167
	9	139	146	147	156	163	170	172	174	169	172
	10	144	151	150	159	167	173	175	179	172	177
	11	148	155	153	163	170	177	179	183	176	181
6	0	153	160	156	166	174	181	183	188	179	186
	1	158	165	160	170	177	184	186	193	182	191
	2	163	169	163	173	181	188	190	197	185	195
	3	167	174	166	177	184	191	194	202	188	200

WOMEN

Height Ft.	ins.	15 yrs.	16—17 yrs.	18—19 yrs.	20—24 yrs.	25—29 yrs.	30—34 yrs.	35—44 yrs.	45—54 yrs.	55—64 yrs.	65 yrs. and over
4	8	96	105	100	106	110	115	126	130	134	120
	9	99	107	103	108	112	117	127	132	137	124
	10	101	110	107	111	114	119	128	134	139	128
	11	104	112	110	113	117	122	130	137	141	132
5	0	107	115	114	116	119	124	131	139	144	136
	1	109	117	118	118	122	126	133	142	146	140
	2	112	120	121	121	124	129	134	144	148	144
	3	115	122	125	123	127	131	135	146	151	148
	4	117	125	129	126	129	133	137	149	153	152
	5	120	127	132	128	132	136	138	151	155	157
	6	123	130	136	131	134	138	140	153	158	161
	7	126	132	140	133	137	140	141	156	160	165
	8	128	135	143	136	139	143	143	158	162	169
	9	131	137	147	138	141	145	144	160	165	173
	10	134	140	151	141	144	147	145	163	167	177
	11	136	142	154	143	146	150	147	165	169	181

dietary limitation from September 1941 to March 1942, showed marked decrease in angina pectoris and myocardial infarction (Brozek, Chapman and Keys [12]). On the other hand, periods of hypertension may follow on recovery from starvation. These authors state that there was virtually an epidemic of hypertension in Leningrad during this period of recovery. An interesting speculation is whether part of this epidemic was not due to the stresses endured by the population which resulted in somatic manifestations that had been masked by starvation. Thus evidence suggests that environment, whether in terms of food or possibly in terms of psychic tension, profoundly influences the reaction of the cardiovascular system.

More direct evidence of improved life expectancy when overweight people reduce their weight is afforded by Fellows's study.[12] This and some later evidence is summarized by Armstrong [10].

While heredity does play an important rôle in developing diabetes mellitus [13], obesity at or prior to onset is one of the most stable characteristics in the medical history of many of these patients. It is interesting to note [14] that annual death rates between 1938 and 1944 from diabetes mellitus fell in a group of German towns

from 23·32 per 100,000 to 15·54, coincidently with a marked deterioration in food supplies.

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MEDICAL ASSOCIATION OF SOUTH AFRICA : OFFICIAL ANNOUNCEMENT

MEDIESE VERENIGING VAN SUID-AFRIKA : AMPTELIKE AANKONDIGING

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held in Red Cross House, 14 Riebeeck Street, Cape Town, on 16, 17 and 18 March 1955, commencing at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
5 February 1955

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word te Red Cross-gebou, Riebeeckstraat 14, Kaapstad, op 16, 17 en 18 Maart 1955, aanvang 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
5 Februarie 1955

ASSOCIATION NEWS : VERENIGINGSNUUS

CLINICAL EVENING OF THE TRANSVAAL PAEDIATRIC SUB-GROUP

A meeting of this Group, in the form of a clinical evening, was held at the Beatrix Branch of the Pretoria Hospital on 13 October 1954. The meeting was well attended and proved most interesting and stimulating; a large number of paediatricians from Johannesburg were present.

Dr. Basil G. van B. Melle presided. The following presented cases:

DR. E. FASSER. (*a*) *Toxoplasmosis.* An Indian child, 2 years old, born and resident in South Africa, presented as a case of mental retardation. The anterior fontanelle was widely open and an air-encephalogram showed atrophy of the cerebral cortex. Serological tests for toxoplasmosis were positive in the mother and child. In both the mother and child the complement fixation was positive, and the Sabin Feldman dye test was positive in a titre of 1 : 256. This is a case of congenital toxoplasmosis and is believed to be the 5th reported case of toxoplasmosis in South Africa, and points to the existence of many more cases in this country which have hitherto been undiagnosed.

(*b*) *Uricaria pigmentosa*, occurring in a European child aged 2¾ years. The lesions appeared for the first time at the age of 9 weeks and continued spreading until at the age of 8 months bullous lesions also developed. Radiographs of the bones did not reveal any abnormalities, but the skin biopsy revealed large accumulation of mast cells in the dermis, particularly in relationship to the small blood vessels. The condition is regarded by many as being a benign mast-cell reticulosis. It was of interest to note that the child showed symptomatic improvement which coincided with administration of vitamin C, vitamin K and rutin.

DR. C. M. ROSS. *Alopecia totalis.* The patient, a girl of 5 years, had developed the condition spontaneously and very rapidly at the age of 2. During treatment with thorium X in 1952 her hair had re-grown to a length of ½ inch, only to fall when applications were discontinued. In 1954 cortisone acetate, in a dose of 10 mg. t.d.s. for 3 months, had produced a similar result, the hair falling when the dose was reduced because of side effects (moon face).

Dr. Ross said that he thought the child should now have a wig and that this should be provided before she was exposed to teasing at school. The meeting agreed that this was safer than continuous long-term cortisone, Dr. van Waalwyk van Doorn mentioning a case where re-growth occurred under a wig after all else had failed.

DR. F. R. JORDAAN. *Du Toni Fanconi Sindroom.* 'n Seun is opgeneem vanweë 'n spontane femur fraktuur. By ondersoek het hy duidelike rachitiese tekens vertoon. In die urine was suiker asook aminosure aanwesig. Bloed-ondersoek toon laë serum fosfor, normale chloriedes, verhoogde alkaliese fosfatase. Sy suster toon 'n identiese beeld. Albei is doofstom, albei toon retinitis pigmentosa, albei is lig van kleur met 'n pofferige vel en hipotoniese spiere. Hulle is tipiese voorbeelde van die sindroom soos beskryf deur du Toni Fanconi.

DR. B. EPSTEIN. *Progressive Spinal Degeneration.* A brother aged 15 years and a sister aged 5 years were presented as unusual types of this condition. They were both mentally retarded and unable to stand or walk without support. Apart from other contractures they both had a remarkably similar degree of flexion contracture of the lateral 3 fingers of each hand. The brother had wasting of the muscles of his limbs with absent tendon reflexes and an extensor plantar response. The sister had an intention tremor, the only sign of a possible cerebellar defect. They were both regressing mentally and physically.

DR. H. P. J. PRETORIUS. (*a*) *Erythroleukaemia* in a boy aged 5 years. Clinically he presented with rapidly developing anaemia and splenomegaly. Peripheral blood showed a leucocyte count rising from 8,000 to 56,000 nucleated cells, with the presence of myeloblasts and erythroblasts. The bone marrow revealed malignancy both of the myeloid and erythroid series, as is typical of the acute type of Erythroleukaemia (Di Guglielmo). Dr. Kingsley demonstrated the slides of this child's blood and bone marrow.

(*b*) *Annular Pancreas.* A premature infant weighing 3 lb. 10 oz. presented with congenital duodenal obstruction. At operation under local anaesthesia this was found to be due to an annular pancreas. There was an associated incomplete rotation of the large bowel. Duodeno-jejunostomy was performed. At present at the age of 6 months the baby appears well and weighs 10 lb.

(*c*) *Waterhouse-Friederichsen Syndrome.* A peripheral blood-smear showing meningococci was demonstrated. This was taken 20 minutes before the death of a 9-months-old baby who presented with the Waterhouse-Friederichsen syndrome and died of meningococcal septicaemia.

DR. L. PANNAL. *Hand-Schuller-Christian Disease*, in a child aged 9 years who presented with acute broncho-pneumonia. At the age of 3 years she had a tumour removed from the left mandible, which on pathological examination proved to be an eosinophilic granuloma.

The positive findings were small stature, underweight, and a pale yellowish complexion. No cutaneous xanthomata were present. She was deaf, with a yellowish discharge from the left ear. She was slightly retarded mentally. Exophthalmos of the left eye was present with enlarged cervical and axillary glands. The spleen was just palpable.

X-ray showed the typical skull appearances. There was absence of the vertical and half of the horizontal ramus of the left mandible, and a large area of erosion particularly of the left base involving the mastoid process and several areas of the right parietal and temporal bones. The lungs presented a picture almost indistinguishable from acute miliary tuberculosis.

The patient was at first treated for the acute lung pathology and later received her first course of deep X-ray therapy to skull and lungs. Clinical improvement was definitely noticed.

PROF. J. G. DAVEL. *Amaurotic Family Idiocy*, in a child first seen at the age of 8 months at the out-patient department of the Pretoria Hospital. His mother thought he could not see but heard very well. His parents are gentiles, first cousins, and both well. There is no family history of a similar affliction. The mother was well during pregnancy, birth was normal, and baby was breast-fed for 6 months.

He could not sit up yet, made strange movements and had a rash all over his body. On examination the hypotonia was striking though the reflexes were all present and equal; for the rest everything was negative except that a cherry-red spot on a greyish white background was seen at the macula on fundoscopic examination.

After 6 weeks' treatment with ACTH the baby was discharged in good physical condition. Seen 9 months subsequently the condition was *in statu quo* except that the baby was now getting occasional fits.

DR. J. G. N. STEYN. *Cooley's Anaemia.* Several cases in one Greek family were demonstrated. Some showed signs of thalassaemis major and some of thalassaemia minor. Repeated blood transfusions were keeping these children alive.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. S. A. Kimmel, M.B., B.Ch. (Rand), D.M.R.D. (London), specialist in Radiological Diagnosis, who has held radiological posts at the Johannesburg Group of Hospitals for 3½ years, has joined Drs. I. A. Brotman and N. Saks in practice at 15 Jenner Chambers, Jeppe Street, Johannesburg.

* * *

Dr. P. W. Hattingh, M.B., Ch.B., Ch.M. (Rand), wat vir 'n jaar in Londen Algemene Chirurgie bestudeer het by die Nagraadse Skool te Hammersmith asook onder Dr. Norman Tanner en vir 'n tyd diens gedoen het te Mile End-hospitaal, Londen, het as Chirurg begin praktiseer te Listergebou, Johannesburg.

* * *

Dr. Maurice Nellen, M.D., M.R.C.P. has resumed practice at 808 Grand Parade Centre, Cape Town.

* * *

The American College of Angiology announces its organization as a scientific body dedicated to the teaching, research and dissemination of knowledge concerned with the science and practice of Angiology. The College is simultaneously assuming responsibility for the publication of the existing periodical 'Angiology' as its official journal. The first official annual meeting of the new College is to be held at Atlantic City, U.S.A., on 4 June 1955. The College also functions as a Specialty Board for the certification of qualified physicians and surgeons as Fellows of the American College of Angiology, and will establish residencies in Angiology in qualified hospitals and medical institutions. The President of the new College of Angiology is Dr. Saul S. Samuels, Editor-in-Chief of the journal 'Angiology', Director of the Department of Angiology and allied Vascular Diseases, Brooklyn Hebrew Hospital for the Aged, and Consulting Vascular Surgeon, Long Beach Memorial Hospital. The headquarters of the College are at 151 East 83 Street, New York City.

* * *

Dr. A. Wolpowitz, M.B., Ch.B. (Cape), D.M.R. (London), has moved his rooms from Central House, Pretoria to The Medical Centre, Pretorius Street, Pretoria, where Dr. J. L. van Rhyn has joined him in practice as Radiologist.

Dr. A. Wolpowitz (Radioloog) het van Sentraalhuis na Mediese Sentrum, Pretoriusstraat, Pretoria verhuis, waar Dr. J. L. van Rhyn saam met hom in praktyk getree het.

* * *

Cape Western Branch. Members are invited to a lecture under the auspices of the University of Cape Town, in the Physiology Lecture Theatre, Medical School, Mowbray, at 8.15 p.m. on Tuesday, 15 February, by Dr. R. S. A. Dean, Director of the Medical Research Council Group for the Study of Infantile Malnutrition at Mulago Medical School, Kampala. Uganda. The title of the Lecture is 'Protein Deficiency in Young Children'.

Dr. Dean is joint author with Drs. Trowell and Davies of the recent monograph on *Kwashiorkor.* He is also the author of a M.R.C. monograph on *Plant Proteins in Child Feeding.*

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis in the period 20 to 27 January 1955:

	European	*Non-European*
Transvaal:		
Johannesburg	12	2
Johannesburg P.U.A.H.B.	2	1
Pretoria	4	
Pretoria P.U.A.H.B.	3	
Lyttelton	1	
Delareyville district	1	
Krugersdorp P.U.A.H.B.	1	1
Vereeniging P.U.A.H.B.	1	
Vereeniging	1	
Rustenburg	1	
Rustenburg district	1	1
Potchefstroom	2	
Andalusia	1	
Boksburg	1	
Germiston	1	
Benoni	1	
Alexandra Health Com.		1
Brakpan		1
Krugersdorp	1	
Groot Marico	1	
Lichtenburg		1
Middelburg		1
Sabie Mun.		1
Total for Transvaal	36	10
Cape Province:		
Sterkstroom		1
Cape Divisional Council	1	1
Matatiele Divisional Council		1
Stellenbosch Divisional Council		1
Middelburg Divisional Council	1	
Vredendal V.M.B.	1	
Qumbu district		1
Total for Cape Province	3	5
Natal:		
Durban	6	2
Durban district	2	
Edenvale		1
Ndwedwe		1
Pinetown		1
Pinetown district		1
Kranskop	1	
Darnall	1	
Port Shepstone district		1
Amanzimtoti	1	
Ladysmith		1
Eshowe district		2
Estcourt		1
Total for Natal	11	11
Orange Free State:		
Bloemfontein		1
Wepener	1	
Frankfort	2 (1 case ex Rhodesia)	
Fauresmith Mun.		1
Total for Orange Free State	3	2
TOTAL FOR THE UNION	**53**	**28**

Union Department of Health Bulletin: Report for the 7 days ended 27 January 1954:

Plague. Orange Free State: No further cases have been reported from the Vredefort district since the notification of 29 December 1954. This area is now regarded as free from infection.

Smallpox: Nil.

Typhus Fever. Orange Free State: One (1) Native case in the Koppies area. Diagnosis confirmed by laboratory tests. Three (3) Native cases in the Harrismith district. Diagnosis of one case confirmed by laboratory tests. The diagnosis of the other cases is based on clinical grounds only.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Dacca, Chalna (Pakistan); Calcutta, Madras (India).

Smallpox in Basra (Iraq); Bombay, Calcutta, Delhi, Jodhpur, Kanpur, Madras (India); Moulmein (Burma); Phnom-Penh (Cambodia); Saigon-Cholon (Viet-Nam).

Typhus Fever: Nil.

CORRESPONDENCE : BRIEWERUBRIEK

SWIMMER'S ITCH

To the Editor: This is in reply to your correspondent A. W. Burton.[1]

In our article we tried to make it quite plain that (1) the itch produced in some subjects by human bilharzial parasites is 'bilharzial itch', (2) the itch produced by the cercariae of non-human parasites is 'swimmer's itch', (3) that all subjects do not react in the same way. Quite apart from the fact that previous sensitization may enhance the reaction, some people are less sensitive than others as in all allergic phenomena. Indeed each of the 3 volunteers in our experiments behaved differently.

Therefore if your correspondent can prove that swimmers may acquire an itch from some other agent, e.g. water mites, then he will have to invent a new term. It will not be 'swimmer's itch'. Bathers may acquire a rash from other organisms in water but almost certainly not from the harmless water mites mentioned. Has Dr. Burton considered the possibility that he is sensitive to pond weeds? Dr. Ordman of this Institute tells me that such a sensitivity is not at all unlikely.

When bathers develop a rash in a bilharzial area two possibilities must be considered, namely bilharziasis and 'swimmer's itch'. If one is prepared to wait 8-10 weeks after the appearance of the rash, in bilharziasis schistosome ova will appear in the urine or faeces provided the patient has acquired a bisexual infection. By that time, of course, some damage has already been done to the liver.[2,3] In 'swimmer's itch' no ova are passed because the infection does not mature. So far our experiments have shown that the two conditions may be differentiated as early as the 3rd week after infection by the 'bilharzia complement-fixation test', which becomes positive in bilharziasis but remains negative in 'swimmer's itch'.

Finally, what makes your correspondent say (3rd paragraph): 'If, as is generally believed and presumably proved . . . .'? Why 'presumably'? Has he any criticisms of the experiments that cause him to doubt the conclusions? If so I should be glad to hear what they are.

Botha de Meillon
South African Institute for Medical Research
Johannesburg
31 January 1955

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